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STUDIES ON A LONG RANGE ASSOCIATION BETWEEN BULBAR POLIOMYELITIS AND PREVIOUS TONSILLECTOMY.

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IN 1952 the writer stated in a paper read at the Eighth Session of the Australasian Medical Congress (British Medical Association) in Melbourne, that the patients who contracted bulbar poliomyelitis during the 1947–1948 poliomyelitis epidemic in South Australia had nearly all undergone tonsillectomy at some time prior to the onset of poliomyelitis, and that in only a few cases was the tonsillectomy recent. An abstract of that paper was published in THE MEDICAL JOURNAL OF AUSTRALIA shortly afterwards (Southcott, 1952). Thus, out of 39 patients with bulbar paralysis, 35[1] had undergone tonsillectomy—a proportion of 90%. Of these 35 tonsillectomized patients with bulbar paralysis, only four had undergone tonsillectomy in the twelve months, and only one in the two months, prior to the onset of poliomyelitis. The preliminary studies suggested that the association between prior tonsillectomy and bulbar poliomyelitis lasted for five to ten years. In the present paper the completed studies of the 1947–1948 bulbar cases are given, and the association and duration are confirmed. The data with regard to the incidence of, and interval since, tonsillectomy are compared with those obtained from a control series of 9000 South Australian school children, made during 1952, and corrected for 1947–1948, together with earlier data on the incidence of previous tonsillectomy in South Australian school children. No association has as yet been shown between previous tonsillectomy and non-bulbar poliomyelitis.

The study has been extended to bulbar cases occurring in South Australia in the period 1949–1950, which confirms the high incidence of previous tonsillectomy in bulbar cases. The data on the interval since tonsillectomy in the 1949–1950 bulbar cases will not be dealt with in this paper, but appear comparable.

Shortly after the publication of the preliminary findings (Southcott, 1952) Top (1952), in the United States of America, published a similar finding of a high incidence (85%) of previous tonsillectomy for a larger North American series of bulbar cases.

In the present paper the completed findings for the 1947–1948 South Australian epidemic will be given. In addition an hypothesis to account for the association will be presented.

HISTORICAL SURVEY.

That tonsillectomy could be a predisposing factor to the contraction of poliomyelitis was first suggested by Sheppard in 1910. The suggestion was renewed by Ayer in 1928, and by Aycock and Luther in 1929. Since then many communications have appeared upon the subject. Attention was focused sharply on the subject by the report of Francis *et alii* (1942) of the disaster at Akron, Ohio, United States of America, in 1942, in which five children out of six in a family were subjected to tonsillectomy, and all five developed bulbar poliomyelitis within nine to fourteen days, three of them dying.

[1] The figures given in 1952 were 34 and five. These have been corrected to 35 and four by further information.

Experimentally there has been confirmation of the predisposing influence of a recent tonsillectomy upon the contraction of poliomyelitis. Thus von Magnus and Melnick (1948) fed poliomyelitis virus to Cynomolgus monkeys immediately after tonsillectomy. This was followed by the contraction of poliomyelitis, though only two monkeys out of the 14 operated on showed bulbar manifestations. Sabin had earlier (1938) demonstrated that the tonsillo-pharyngeal inoculation of poliomyelitis virus into monkeys resulted in a high incidence of bulbar poliomyelitis. In 13 cases out of 17 this was the presenting symptom. These findings have been extended by the work of Faber *et alii* (1951) in a series of experiments in which poliomyelitis virus was applied to the pharynx of Cynomolgus monkeys, and tonsillectomy was then performed. In all these seven monkeys bulbar poliomyelitis followed within seven to ten days, associated in three monkeys with a high spinal (cervical) paralysis. Control experiments in which oropharyngeal swabbing with virus was carried out, but without tonsillectomy, in seven monkeys resulted in five of these contracting poliomyelitis, one showing bulbar paralysis and the others spinal paralysis.

In general there has been a recognition that recent tonsillectomy increases the risk of contracting bulbar paralysis during poliomyelitis epidemics, and this has been reflected in public health administration. Anderson (1945) showed that in Utah, United States of America, in 1943, tonsillectomy within the previous thirty days predisposed the subject to bulbar poliomyelitis. Seventeen cases out of 39, or 43%, were preceded by tonsillectomy within the thirty days prior to the onset. The incidence of poliomyelitis in recently tonsillectomized children was found to be 2·6 times that in the general child population. For bulbar poliomyelitis the ratio was 16 : 1. An important communication on the subject from an epidemiological aspect has come from Siegel *et alii*, who have shown (1951*a*) that in the 1949 epidemic in New York City recent tonsillectomy predisposed the subject to poliomyelitis. This influence was statistically significant for one month after tonsillectomy. The results were "possibly significant" for as long as three or four months after tonsillectomy. Within one month after tonsillectomy the risk appeared to be several times greater than for others of comparable age in the general population. In a second paper the same authors (1951*b*) showed that in New York City over the period 1944 to 1949 the percentage of bulbar paralysis was significantly higher than was expected among tonsillectomized patients within one month of operation. The results were inconclusive from one to six months after operation, but there was no evidence that tonsillectomy exerted any influence in the six to twelve months period after tonsillectomy. In this period 1944 and 1949 were epidemic years in New York City, and the years 1945 to 1948 were non-epidemic.

On the other hand, some epidemiological investigators have not been able to find any connexion between poliomyelitis and recent tonsillectomy—for example, Cunning (1947), Miller (1951). However, the methods of these investigators are open to criticism. Thus Cunning's technique of gathering data has been criticized as allowing so much dilution of the data that any connexion, if present, would not be demonstrated (Wilson, 1952; Faber, 1949).

Reviews of the subject are to be found in the articles by Siegel *et alii* (1951 *a* and *b*), by Aycock (1942), by Glover (1948), by van Rooyen and Rhodes (1948), and in *The Lancet* (Editorial, 1949). However, these findings relate to recent tonsillectomy only, of the duration of one month or so, and not to the long-term association between bulbar poliomyelitis and previous tonsillectomy discovered independently by the present writer and by Top. Top in his article (1952) presents the data on the intervals that have elapsed between tonsillectomy and poliomyelitis, but does not analyse them further or draw any conclusions on the duration of the association between them, on account of lack of control data on tonsillectomy. In the present study statistical evidence will be presented, in which the association in the South Australian cases will be traced for at least nine to ten years.

PRESENT INVESTIGATION.

The present investigation arose out of a reexamination during 1952 by the present writer of some of the data gathered in an epidemiological and clinical survey conducted together with N. D. Crosby in 1947–1948 on the epidemic of poliomyelitis then occurring in South Australia (see Southcott and Crosby, 1949). That epidemic had a high incidence of bulbar forms among the paralytic cases—40 out of a total 103 cases, or 39%. From that survey data were available on 78 out of the 103 paralysed patients as to whether they had undergone tonsillectomy, but were not analysed at the time. Examination showed that of these 78 patients, 44 had undergone tonsillectomy and 34 had not. This proportion of 44 in 78 or 56% appeared to the present writer to be a high proportion to have undergone tonsillectomy, although no control data were available. As was recorded earlier (Southcott and Crosby, 1949), there was only one patient in the whole series who had had a tonsillectomy performed in the six weeks prior to the onset of the disease; this was a girl, aged ten years, in a country town, who had contracted bulbar poliomyelitis, with the onset ("E.P.D." or "earliest prodromal date") fifteen days after tonsillectomy. It therefore appeared that any association between poliomyelitis and previous tonsillectomy in that epidemic, if confirmed by further investigation, was likely to be a long range one. Such a phenomenon had not been recorded previously.

It was accordingly decided to conduct a further investigation with the following objects: (i) to complete the data for the remaining 25 paralytic cases of the 1947–1948 poliomyelitis epidemic as to whether tonsillectomy had been performed or not, and if so, when; (ii) to obtain a control series of similar data on the general population of South Australia, for comparison; (iii) to analyse the tonsillectomy data of the poliomyelitis cases by age and clinical type.

Subsequently the investigation was extended to the bulbar cases of the 1949–1950 epidemic of poliomyelitis in South Australia.

Collection of Data of the Poliomyelitis Cases.

It was decided to investigate further the 103 paralytic cases of the 1947–1948 epidemic, and not to investigate further the 60 non-paralytic cases revealed by the earlier survey, on the following grounds: (i) The diagnosis of poliomyelitis was reasonably certain in the former, being confirmed by clinical and epidemiological study, and by an extensive pathological study of post-mortem material (Fowler, 1951); moreover, these patients or their relatives were aware of the diagnosis, which was often not the case with the non-paralytics. (ii) It was considered that the 103 paralytic cases comprised practically the total population of the paralytic cases occurring between July 1, 1947, and June 30, 1948. This statement is made on account of the exhaustive inquiries made during the epidemic into any suspected case of poliomyelitis, together with the acute awareness of the medical profession and the public of the presence of a poliomyelitis epidemic, enhanced by a good deal of publicity about the disease in the daily Press. On the other hand, many non-paralytic cases must have escaped attention.

Accordingly, after a few replies had been received by telephone from the conveniently situated metropolitan patients in Adelaide, the following letter and circular *questionnaire* were sent out for the remaining 20 or so patients, in March, 1952.

Dear Sir/Madam,

I should be extremely grateful if you could send me as soon as possible, the following information with regard to the patient/yourself.

The answers that you give may help in the study of poliomyelitis, and I know that I can be sure of your willingness to help. Naturally, the replies will be treated as strictly confidential.

Yours sincerely, etc.

Serial Number..... F1
Name of Patient.....
Date of Birth.....Day.....Month 19.....
Have the patient's Tonsils been removed? .....
When were the Tonsils removed .....Day.....Month 19....
(Give date as accurately as possible)
By which Doctor?.....
Where? .....
Has the patient suffered from Chickenpox?.....
State date of Chickenpox as accurately as possible.....
Has the patient's Appendix been removed?.....
Give date Appendix removed as accurately as possible.....
By which Doctor?.....
Where? .....

Could you please return this *questionnaire* sheet, as soon as possible, to (etc.).

These sheets were sent out for the available remaining paralytic cases. The questions about chicken-pox and appendicectomy were put in so as not to draw too much attention to tonsillectomy, and possibly to form some kind of control. From only four patients out of the 103 paralytics was no reply finally received. One of these four had had paralysis of bulbar type. In addition

it was known that another of these patients who did not reply, who had had non-bulbar paralysis, had undergone tonsillectomy prior to the contraction of poliomyelitis, although the interval was not known. Thus the required data were available for 99 out of 103 cases, or 96%. One reply was received from as far away as the New Hebrides, whence the *questionnaire* was forwarded. This high percentage response was very gratifying, particularly as in most cases four years had elapsed since the contraction of poliomyelitis.

Definitions of Clinical Types of Poliomyelitis.

As in the earlier study (Southcott and Crosby, 1949), poliomyelitis is divided into non-paralytic and paralytic types. The paralytic cases were divided in the earlier study into three clinical types: (i) encephalitic, (ii) bulbar, (iii) spinal. Any one or more of these forms could be present in an individual case. The cases were classified thus four years before, prior to any appreciation of the possible significance of tonsillectomy. A reexamination of the clinical histories showed no case that was classified differently. The term "encephalitic" as used with these cases means cerebral or mid-brain involvement, and a case was so classified if any of the following was shown: (*a*) irrationality (not secondary to terminal anoxia); (*b*) diplopia from an oculomotor lesion; (*c*) coma in the absence of respiratory paralysis. Bulbar poliomyelitis was diagnosed if any of the following was present: (*a*) difficulty in swallowing; (*b*) nasal regurgitation of food or fluid; (*c*) nasal tone and slurring of speech, not encephalitic in origin; (*d*) pooling of mucus in the oro-pharynx or naso-pharynx; (*e*) primary vasomotor collapse; (*f*) respiratory paralysis not considered due to a spinal cord lesion; (*g*) diplopia due to an abducens lesion; (*h*) facial weakness or paralysis. Nystagmus was present in several of the bulbar cases, and considered as indicative of a vestibular lesion, but it was not used in this series as a sole criterion, as other evidence of bulbar paralysis as given above was present. Spinal poliomyelitis was diagnosed if there was muscle paralysis or weakness. In this paper the paralytic cases are divided into bulbar and non-bulbar on the criteria given above.

TABLE I.

Incidence of Previous Tonsillectomy in the 103 Paralytic Cases of the 1947-1948 South Australian Epidemic of Poliomyelitis.

Rural or Urban.	Tonsillectomy.			Total.
	Previously Performed.	Not Previously Performed.	Not Known.	
(a) As Known from the Survey of 1947–1948.				
Rural	23	15	10	48
Urban	21	19	15	55
Total ..	44	34	25	103
(b) As Revealed by the 1952 Survey.				
Rural	7	1	—	8
Urban	5	9	—	14
Total	12	10	—	22
(c) As Known After the 1952 Survey.				
Rural	30	16	2	48
Urban	26	28	1	55
Total	56	44	3	103

The term "non-paralytic poliomyelitis" is used for any case of poliomyelitis without objective evidence of neurological involvement. It includes the term "abortive" of some authors.

More extensive clinical classifications for poliomyelitis are given by some authors—for example, Wickman (1907, 1913), who recognized eight groups, or Powell (1938), who recognized six, three of which would here be considered as "non-paralytic". The classification adopted above was adopted for purposes of simplification, and appears to be in conformity with modern trends (see van Rooyen and Rhodes, 1948).

The most extensive study of the mechanism and pathology of bulbar poliomyelitis in recent years is due to Baker (1949) and to Baker *et alii* (1950). The only difference between the criteria given and Baker's is that he classifies oculomotor lesions within bulbar forms. In the present paper such lesions are classified with cerebral lesions under the term "encephalitic". However, these oculomotor lesions do not form a large element, being present in only 11% of Baker's 183 cases. In the present series oculomotor lesions were seen occasionally in conjunction with lesions that were more strictly "bulbar". In the present series in practically all of the bulbar cases (35 out of 40 cases) difficulty in swallowing or phonation was present, indicating a lesion of the *nucleus ambiguus*.

Incidence of Previous Tonsillectomy in Paralytic Poliomyelitis: The Results of the Surveys on the 1947-1948 Poliomyelitis Cases.

From the earlier data it was known that 44 out of 78 paralysed patients had previously undergone tonsillectomy; after the 1952 survey it was known that 56 out of 100 paralysed patients had previously undergone tonsillectomy. These figures need to be tested for homogeneity. They are set out in Table I, divided into urban and rural cases.

Testing firstly for the homogeneity of the proportion of the previously tonsillectomized among rural and urban paralytic patients, and ignoring the "Not Known" class, we have the following figures from Table I (*a*):

23	15	38
21	19	40
44	34	78

Thus $\chi_c^2 = 0 \cdot 236$ on one degree of freedom, which is not significant. Similarly, from Table I (*c*) we have the following figures:

30	16	46
26	28	54
56	44	100

Thus $\chi_c^2 = 2 \cdot 29$ on one degree of freedom, which is not significant.

Thus homogeneity is not contradicted, and we may pool the rural and urban classes, as is done in Table I (*c*). Testing for homogeneity with these pooled classes (ignoring the "Not Known" class as being now too small to influence significance), we have the following figures (Table II):

TABLE II.

Investigation.	Tonsillectomy.		Total.
	Previously Performed.	Not Previously Performed.	
Investigation of 1947–1948	44	34	78
Investigation of 1952 ..	12	10	22
Total	56	44	100

Here $\chi^2 = 0 \cdot 0242$ on one degree of freedom, which is not significant.

We may therefore pool the total figures from the surveys. It will be noted that 56 out of 100 patients with paralytic poliomyelitis, or 56%, had previously undergone tonsillectomy.

Analysis of Crude Data of the Proportion Tonsillectomized in Control South Australian School Children.

An analysis of the age incidence of the 1947–1948 South Australian poliomyelitis cases was given earlier (Southcott and Crosby, 1949). School children contribute a large proportion to

a poliomyelitis epidemic, and data on school children may be used as a control. An inquiry of Dr. W. Christie, Principal Medical Officer of the Schools Medical Services of South Australia, revealed that data on the numbers of school children previously tonsillectomized were available for past years in crude form, not divided by age or sex. These data were derived from answers given by parents on form MB21 to a number of routine questions about the health and past medical history of each child. (Later these forms, after some modification, were used to get data on the interval since tonsillectomy *et cetera*.) About 20,000 school children are examined annually by the medical officers of the Schools Medical Services. The children are examined in Grades I, IV and VII, corresponding roughly to ages six, nine and twelve years; also those still at school are examined in later grades, corresponding roughly to ages fourteen and sixteen years. Consolidated data with regard to previous tonsillectomy are shown in Table III.

TABLE III.

Data on Incidence of Previous Tonsillectomy in School Children Surveyed by the Schools Medical Services of South Australia.

Year.	Number of Children Examined.	Number Previously Tonsillectomized.	Percentage Rate.
1940	15,627	2906	18·60
1941	14,738	2781	18·87
1942	15,915	3135	19·70
1943	14,888	3225	21·66
1944	12,647	3018	23·86
1945	10,905	2899	26·58
1946	14,284	4404	30·83
1947	24,627	6510	26·43
1948	21,236	5906	27·81
1949	18,888	5612	29·71
1950	22,077	6269	28·40
1951	27,080	7289	26·92

Testing the proportions shown for 1947 and 1948 for homogeneity, we find $\chi^2=10{\cdot}98^{**}$ on one degree of freedom, $P<0{\cdot}01$.[1] The figures show an increase for 1948 which is significant at the 1% level.

Testing for homogeneity the proportions shown for 1946, 1947 and 1948 similarly, we find $\chi^2=87{\cdot}2^{***}$ on two degrees of freedom, $P<0{\cdot}001$. The test shows a highly significant lack of homogeneity.

With regard to the 1947 and 1948 figures, one could reasonably expect to find a decrease in the proportion of the tonsillectomized, rather than an increase, on account of the widely publicized (and followed) recommendations against the performance of tonsillectomy during the poliomyelitis epidemic. The lack of homogeneity shown can presumably be accounted for by the sampling technique being not uniform. This hypothesis will next be tested.

TABLE IV.

For the Year 1947.

Urban or Rural.	Tonsillectomy.		Total.	Percentage of Subjects Tonsillectomized.
	Previously Performed.	Not Previously Performed.		
Urban	4010	9487	13,497	24·78
Rural	2500	8630	11,130	22·42
Total	6510	18,117	24,627	

Here $\chi^2=164{\cdot}8^{***}$ on one degree of freedom, and $P<0{\cdot}001$.

Unpublished departmental figures were made available by Dr. Christie. These indicated that the sampling technique was not uniform. It had not been possible to examine each school each year, it being necessary in some years to concentrate the medical surveys to cover particular parts of the State. The figures were available for each school as a total, but not divided further by age, sex *et cetera*. Allotting these figures for 1947 and 1948 by rural and urban distribution,[1] we have the data for scholars examined (Tables IV and V).

Hence for 1947 the proportion of the previously tonsillectomized was significantly higher in the urban than in the rural scholars examined.

TABLE V.

For the Year 1948.

Urban or Rural.	Tonsillectomy.		Total.	Percentage of Subjects Tonsillectomized.
	Previously Performed.	Not Previously Performed.		
Urban	4078	9109	13,187	30·96
Rural	1828	6221	8,049	22·74
Total	5906	15,330	21,236	

Here $\chi^2=167{\cdot}9^{***}$ on one degree of freedom, and $P<0{\cdot}001$.

Here again we have found that the proportion of the previously tonsillectomized is significantly higher in the urban than in the rural scholars examined. (It may be remarked here that the 1952 survey of scholars has shown that the proportion of the previously tonsillectomized is consistently higher in the urban scholars—see Tables XXXI to XXXIV and Figures I and II.)

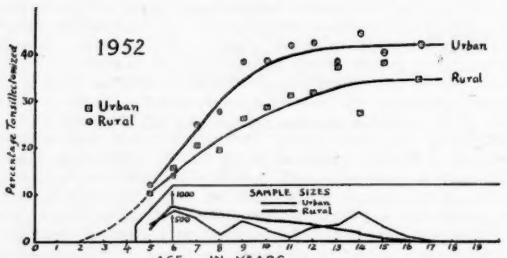

FIGURE I.

Graphs to show proportions of previously tonsillectomized in South Australian school children by annual age groups, as revealed by a survey of the data for 9052 scholars in 1952. The inset diagram shows sample sizes.

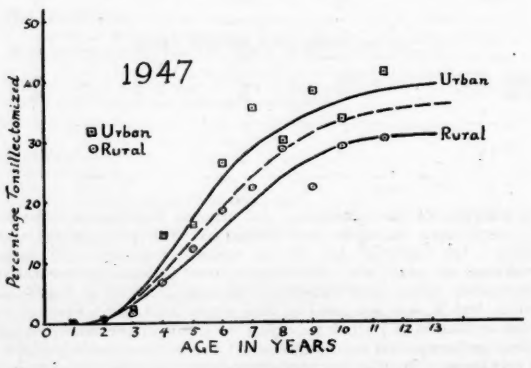

FIGURE II.

Graphs to show the proportion of previously tonsillectomized for South Australian school children by annual age groups, for 1947. Data calculated from the results of the 1952 survey.

[1] $\chi^2 = 10{\cdot}98^{**}$ means that this is highly significant. One asterisk would mean "significant".

[1] In this article the terms rural and urban are used as in the earlier survey (Southcott and Crosby, 1949) synonymously with extrametropolitan and metropolitan. The Schools Medical Services figures have been corrected to this sense.

Testing over the years 1947 and 1948 the proportions tonsillectomized in the urban and rural scholars separately, we have the figures shown in Tables VI and VII:

TABLE VI.
Urban (Metropolitan) Scholars.

Year.	Tonsillectomized.	Not Tonsillectomized.	Total.	Percentage Tonsillectomized.
1947	4010	9487	13,497	24·78
1948	4078	9109	13,187	30·96
Total ..	8088	18,596	26,684	

Here $\chi^2=4\cdot65^*$ on one degree of freedom and $P<0\cdot05$.

TABLE VII.
Rural (Extra-Metropolitan) Scholars.

Year.	Tonsillectomized.	Not Tonsillectomized.	Total.	Percentage Tonsillectomized.
1947	2500	8630	11,130	22·42
1948	1828	6221	8,049	22·74
Total ..	4328	14,851	19,179	

Here $\chi^2=0\cdot166$ on one degree of freedom, which is not significant.

It is thus found that for 1947 and 1948 the proportions of the previously tonsillectomized in the rural scholars examined are homogeneous, no significant change being present. For the urban scholars examined the proportions are not homogeneous. This lack of homogeneity could be explained by sampling differences. It must be realized that the figures given are for crude samples, not standardized by age distribution *et cetera*. In support of this argument the considerable differences in age distributions in the samples of children examined in 1952 will be shown later.

Despite these differences, it is nevertheless a legitimate procedure to pool the 1947 and 1948 figures for comparison with similar data for the poliomyelitis cases of the scholar class of the 1947–1948 epidemic.

The Association between Previous Tonsillectomy and Paralytic Poliomyelitis: Preliminary Demonstration of This in Scholars by Comparison with the Crude Data for Control School Children.

It is proposed to consider here the proportion of the previously tonsillectomized among scholars in the 1947–1948 paralytic poliomyelitis series, and to compare this with the figures given for the scholars surveyed by the Schools Medical Services of South Australia in the years 1947 and 1948. Although much of what is considered in this section will be amplified later from data obtained from the 1952 survey of school children, and the comparisons made are to a certain extent vitiated by sampling variations for the school children (mainly for age distribution), yet the association is so pronounced that it is possible to demonstrate it from the crude figures. The data given here give a basis for comparison without the complicated detail of standardization which will be necessary later. Furthermore, control data for various age groups for the years 1947 and 1948 can be reconstructed only partially from the data of the 1952 scholar survey.

Subjects of paralytic poliomyelitis of the 1947–1948 epidemic were classified as "scholars" if they were still attending primary or secondary school at the time of contraction of the disease (although, for example, if they were on vacation they were still classed as "scholars"). Table VIII shows data on previous tonsillectomy for 1947–1948 paralytic poliomyelitis cases, divided into urban and rural classes, and into scholars and non-scholars.

TABLE VIII.
Data on Numbers Previously Tonsillectomized in 1947–1948 Cases of Paralytic Poliomyelitis, divided into Scholars and Non-scholars, by Rural and Urban Distribution.

Subjects.	Tonsillectomized.	Not Tonsillectomized.	Not Known.	Total.
(*a*) Rural.				
Scholars	18	6	1	25
Non-scholars ..	12	10	1	23
Total	30	16	2	48
(*b*) Urban.				
Scholars	17	8	0	25
Non-scholars ..	9	20	1	30
Total ..	26	28	1	55
(*c*) Rural and Urban Combined.				
Scholars	35	14	1	50
Non-scholars ..	21	30	2	53
Total	56	44	3	103

Testing the homogeneity of the proportions of the previously tonsillectomized among the rural and urban paralytic poliomyelitis cases, we have the following figures (Table IX):

TABLE IX.
Scholars.

Scholars.	Tonsillectomized.	Not Tonsillectomized.	Total.
Rural ..	18	6	24
Urban ..	17	8	25
Total ..	35	14	49

Here $\chi_c^2=0\cdot051$ on one degree of freedom, which is not significant.

TABLE X.
Non-Scholars.

Non-Scholars.	Tonsillectomized.	Not Tonsillectomized.	Total.
Rural ..	12	10	22
Urban ..	9	20	29
Total ..	21	30	51

Here $\chi_c^2=1\cdot97$ on one degree of freedom, which is not significant.

TABLE XI.

Subjects.	Tonsillectomized.	Not Tonsillectomized.	Total.
Scholars ..	35	14	49
Non-scholars ..	21	30	51
Total ..	56	44	100

Here $\chi_c^2=8\cdot10^{**}$ on one degree of freedom, and $P<0\cdot01$.

We may therefore pool rural and urban subjects in each case as is done in Table VIII (c).

Testing whether the proportion of tonsillectomized subjects differs between scholars and non-scholars, we have, amalgamating rural and urban, the figures shown in Table XI.

We may conclude from this that in the cases of paralytic poliomyelitis the proportion of the tonsillectomized is significantly higher, at the 1% level of probability, in scholars than in non-scholars. Testing rural and urban figures separately, we have the figures shown in Tables XII and XIII.

TABLE XII.
Rural Figures.

Subjects.	Tonsillectomized.	Not Tonsillectomized.	Total.
Scholars ..	18	6	24
Non-scholars ..	12	10	22
Total ..	30	16	46

Here $\chi^2_c = 1 \cdot 31$ on one degree of freedom, which is not significant.

TABLE XIII.
Urban Figures.

Subjects.	Tonsillectomized.	Not Tonsillectomized.	Total.
Scholars ..	17	8	25
Non-scholars ..	8	21	29
Total ..	25	29	54

Here $\chi^2_c = 5 \cdot 94^*$ on one degree of freedom, and $P < 0 \cdot 02$.

It appears, then, that the urban figures are responsible for the lack of homogeneity between scholars and non-scholars noted above.

It will be noted that 35 out of 49 scholars with paralytic poliomyelitis had been tonsillectomized, a proportion of 71%. Testing whether the proportions of the tonsillectomized differ between scholars of the paralytic series and those surveyed by the Schools Medical Services of the Education Department in 1947–1948, we have the figures shown in Table XIV.

TABLE XIV.

Subjects.	Tonsillectomized.	*Not* Tonsillectomized.	Total.
Scholars, poliomyelitis series	35	14	49
Scholars, *control*, Education Department ..	12,416	33,447	45,863
Totals	12,451	33,461	45,912

Here $\chi^2 = 48 \cdot 77^{***}$ on one degree of freedom, and $P < 0 \cdot 001$.

Thus it may be concluded that the probability is less than 0·1% that the proportions of the tonsillectomized are the same in the two populations considered. Now if (i) the Education Department figures are characteristic of the whole population of scholars, and (ii) all the paralytic poliomyelitis cases are included in the series described, or else the series is a random sample in every respect, then we may conclude that paralytic poliomyelitis is more likely to occur among scholars who have been tonsillectomized than among those who have not.

With regard to the first criterion, certain objections may be raised. For example, the Education Department of South Australia does not control all school children in the State, there being a number of private schools. However, these do not comprise a large proportion of scholars, nor was there a large proportion of scholars from private schools in the poliomyelitis series, nor does there appear any reason why the proportion of tonsillectomized among these scholars should differ greatly from scholars attending Education Department schools. A more important objection could be raised in the possibility of considerable sampling variation in the scholars surveyed by the Schools Medical Services of the Education Department, which has already been suggested. Nevertheless, the highest proportion recorded of scholars tonsillectomized in any of the years shown in Table III is 30·8% (for 1946), which is well below the observed proportion for scholars with paralytic poliomyelitis of 35 out of 49 or 71%, the difference being significant.

With regard to the second criterion, it is considered that this presents no difficulty. Reasons were given earlier for believing that the paralytic series studied comprised practically the total population of paralytic cases for the State between the times specified.

It is considered, therefore, that the tests shown above indicate that there was a higher proportion of the previously tonsillectomized among scholars with paralytic poliomyelitis than among controls. It will later be shown that association between the contraction of paralytic poliomyelitis and previous tonsillectomy was due mainly or entirely to the large number of bulbar cases in the paralytic series. Standardization of data by age groups will be dealt with later, particularly in reference to the duration of the association shown.

Bulbar Incidence and Fatality Rates in the 1947–1948 Epidemic.

Noteworthy features of the 1947–1948 South Australian epidemic were the high bulbar incidence and the high death rate. These data are analysed in Table XV.

TABLE XV.
Incidence of Bulbar Paralysis and Fatality Rate of the 103 Paralytic Cases of the 1947–1948 South Australian Epidemic.

Rural or Urban.	Fatal or Non-Fatal.	Bulbar.	Non-Bulbar.	Total.
(a) Divided into Rural and Urban Cases.				
Rural ..	Fatal	6	2	8 } 48
	Non-fatal ..	13	27	40 }
Urban ..	Fatal	10	2	12 } 55
	Non-fatal ..	11	32	43 }
Total ..	.. .. ..	40	63	103
(*b*) Rural and Urban Cases Combined.				
Rural *plus* urban ..	Fatal	16	4	20
	Non-fatal ..	24	59	83
Total ..	.. .. ..	40	63	103

From Table XV (*b*) we calculate $\chi^2_c = 15 \cdot 62$ on one degree of freedom, $P < 0 \cdot 001$. Calculating the exact probability, we find that $P = 0 \cdot 0000530$.

It is thus shown that there is a highly significant association between incidence of bulbar paralysis and fatality. It will be noted that the incidence of bulbar involvement in the paralytic series was 40 out of 103, or 39%. Of the 40 patients with bulbar paralysis 16 died, an incidence of 40%; of the 63 non-bulbar paralytics only four died, an incidence of 6·4%. Of the 20 deaths of the series 16 occurred in bulbar cases; that is, 80% of the deaths were in the bulbar cases.

The Association between Previous Tonsillectomy and Bulbar Poliomyelitis.

It is proposed in this section to divide the 1947–1948 paralytic cases into bulbar and non-bulbar forms, and to compare the

proportions of the previously tonsillectomized subjects. This analysis is set out in Table XVI.

TABLE XVI.

Analysis of 1947–1948 Paralytic Poliomyelitis into Bulbar and Non-Bulbar Forms, Compared with the Numbers of Tonsillectomized and Non-Tonsillectomized Subjects.

Subjects.	Bulbar Paralysis.	Non-Bulbar Paralysis.	Total.
(a) Rural Cases.			
Tonsillectomized	17	13	30
Not tonsillectomized ..	1	15	16
Not known	1	1	2
Total	19	29	48
(b) Urban Cases.			
Tonsillectomized	18	8	26
Not tonsillectomized ..	3	25	28
Not known	0	1	1
Total	21	34	55
(c) Rural and Urban Cases Combined.			
Tonsillectomized	35	21	56
Not tonsillectomized ..	4	40	44
Not known	1	2	3
Total	40	63	103

The homogeneity of rural and urban populations with regard to the distribution of bulbar and non-bulbar forms between tonsillectomized and non-tonsillectomized patients is evident from inspection, hence parts (*a*) and (*b*) of Table XVI may be amalgamated as is done in part (*c*) of Table XVI. In Table XVI (*c*), if we ignore the "Not Known" class temporarily, we may test the proportions shown thus (Table XVII):

TABLE XVII.

Subjects.	Bulbar Paralysis.	Non-Bulbar Paralysis.	Total.
Tonsillectomized	35	21	56
Not tonsillectomized ..	4	40	44
Total	39	61	100

$\chi_c^2 = 27 \cdot 35^{***}$ on one degree of freedom, $P < 0 \cdot 001$.

Now if we allot the "Not Known" class to reduce significance as far as possible, we have the following figures:

35	23	58
5	40	45
40	63	103

Here $\chi_c^2 = 23 \cdot 8^{***}$ on one degree of freedom, and P is still less than $0 \cdot 001$.

It may therefore be concluded that the probability of obtaining results as discrepant as this or more discrepant on the hypothesis that the number of subjects with bulbar and non-bulbar paralytic poliomyelitis contains the same proportion of the previously tonsillectomized is less than one in 1000.

It has now been shown that in the 1947–1948 epidemic in South Australia a previous tonsillectomy was associated with (i) the contraction of paralytic poliomyelitis and (ii) the contraction of the bulbar form of paralytic poliomyelitis. The question then arises whether association (i) is due to (ii), or whether the effects are at all separate. This will next be tested. In order to make valid comparisons with controls the data will be divided into those for scholars and non-scholars, as the only reliable figures for controls are for scholars. Table XVIII shows the data for the paralytic cases divided for scholars and non-scholars, bulbar and non-bulbar paralysis, tonsillectomized and non-tonsillectomized subjects, rural and urban distribution.

TABLE XVIII.

Data on 1947–1948 Paralytic Cases, divided for Bulbar and Non-Bulbar Forms, for Scholars and Non-Scholars, by Rural and Urban Distribution.

Subjects.	Paralysis.	Tonsillectomy. Previously Performed.	Tonsillectomy. Not Previously Performed.	Tonsillectomy. Not Known.	Total.
(a) Rural Cases.					
Scholars ..	Bulbar ..	13	0	1	14
	Non-bulbar	5	6	0	11
Non-scholars	Bulbar ..	4	1	0	5
	Non-bulbar	8	9	1	18
Total ..	..	30	16	2	48
(b) Urban Cases.					
Scholars ..	Bulbar ..	14	1	0	15
	Non-bulbar	3	7	0	10
Non-scholars	Bulbar ..	4	2	0	6
	Non-bulbar	5	18	1	24
Total ..	.. ..	26	28	1	55
(c) Rural and Urban Cases Combined.					
Scholars ..	Bulbar ..	27	1	1	29
	Non-bulbar	8	13	0	21
Non-scholars	Bulbar ..	8	3	0	11
	Non-bulbar	13	27	2	42
Total ..	.. ..	56	44	3	103

Rural and urban populations in the various classes are evidently homogeneous with respect to proportion of the previously tonsillectomized, except possibly for non-scholars with non-bulbar paralysis. Testing for homogeneity for this class, we have the following figures (Table XIX):

TABLE XIX.

Rural or Urban.	Tonsillectomized Subjects.	Non-Tonsillectomized Subjects.	Total.
Rural ..	8	9	17
Urban ..	5	18	23
Total ..	13	27	40

Thus $\chi_c^2 = 1 \cdot 82$ on one degree of freedom, which is not significant.

We may therefore amalgamate the rural and urban figures as is done in Table XVIII (*c*).

Testing the homogeneity of the proportions of previously tonsillectomized subjects in bulbar and non-bulbar paralysis classes, and ignoring the "Not Known" class, we have, for scholars, the figures shown in Table XX.

Performing an exact test we find that $P = 0 \cdot 0000084$; that is, in the class of scholars with paralytic poliomyelitis a significantly higher proportion of the bulbar form occurs among those previously tonsillectomized than among those not previously tonsillectomized.

TABLE XX.

Paralysis.	Tonsillectomized Scholars.	Non-Tonsillectomized Scholars.	Total.
Bulbar ..	27	1	28
Non-bulbar	8	13	21
Total ..	35	14	49

Testing the homogeneity of the proportions of the previously tonsillectomized among scholars in the bulbar poliomyelitis class with the proportion of scholars surveyed by the Schools Medical Services in the period 1947–1948, we have the figures set out in Table XXI.

TABLE XXI.

Subjects.	Tonsillectomized.	Not Tonsillectomized.	Total.
Bulbar poliomyelitis scholars	27	1	28
Education Department scholars	12,416	33,447	45,863
Total	12,443	33,448	45,891

Performing an exact test, we find that $P=1\cdot015\times10^{-14}$.

We may therefore conclude that the proportion of the previously tonsillectomized is significantly greater in the bulbar poliomyelitis class than among controls.

Testing the homogeneity of the proportions of the previously tonsillectomized among scholars in the non-bulbar paralytic class with the proportion for the scholars surveyed by the Schools Medical Services for 1947–1948, we have the figures shown in Table XXII.

TABLE XXII.

Subjects.	Tonsillectomized.	Not Tonsillectomized.	Total.
Non-bulbar poliomyelitis scholars	8	13	21
Control scholars, Education Department ..	12,416	33,447	45,863
Total	12,424	33,460	45,884

Thus $\chi_c^2=0\cdot794$ on one degree of freedom, which is not significant; that is, the data do not contradict the hypothesis that the proportions are the same.

It may be concluded that for the figures at present available the bulk of the association of previous tonsillectomy with paralysis in scholars is due to the association of previous tonsillectomy with bulbar poliomyelitis. No evidence has been found of an association between previous tonsillectomy and non-bulbar paralytic poliomyelitis in scholars.

Testing the proportion of the previously tonsillectomized in non-scholars, we have the figures shown in Table XXIII.

Ignoring the "Not Known" class, and testing the homogeneity of the proportions eight out of 11 and 13 out of 40, we find that $\chi_c^2=4\cdot22$* on one degree of freedom, which is a significant result as $P<0\cdot05$. Calculating the exact probability, we find that $P=0\cdot035$. It has therefore been shown that there is a significantly higher proportion of the previously tonsillectomized in the bulbar as against the non-bulbar paralytic class in non-scholars. The non-scholars are made up principally of the older age groups, from the age of fourteen onwards. The finding in non-scholars indicates an extension of the association between previous tonsillectomy and bulbar poliomyelitis, which will be dealt with further in the consideration of the duration of the association.

TABLE XXIII.

Type of Poliomyelitis.	Tonsillectomized Subjects.	Non-Tonsillectomized Subjects.	Not Known.	Total.
Bulbar poliomyelitis	8	3	0	11
Non-bulbar paralytic poliomyelitis ..	13	27	2	42
Total	21	30	2	53

Final Demonstration of the Association between Bulbar Poliomyelitis and Previous Tonsillectomy, and the Lack of Association with Non-bulbar Paralytic Poliomyelitis, in the 1947–1948 South Australian Epidemic, by the Use of Data Standardized for Age Distribution.

So far we have needed to use the crude data on Education Department scholars as our controls, unstandardized by age distribution. In order to maintain continuity it is proposed to consider here standardized data, corrected to 1947–1948, derived from later surveys. The data with regard to the age distributions of bulbar and non-bulbar paralytic poliomyelitis have been taken from smoothed curves derived from the figures shown in Table XXVI. The percentage of previously tonsillectomized for each age group is derived from the middle curve drawn in Figure II. The process of obtaining standardized proportions is shown in Table XXIV.

TABLE XXIV.

Table to Show Expected Proportions of Previously Tonsillectomized Subjects by Each Annual Age Group, for Combined (Rural and Urban) Population of South Australia, 1947–1948.

A. Age. (Years.)	B. t.[1]	Bulbar Poliomyelitis. C. f.[2]	Bulbar Poliomyelitis. D. ft.[3]	Non-Bulbar Paralytic Poliomyelitis. E. f.	Non-Bulbar Paralytic Poliomyelitis. F. ft.
0	0·0	0·00	0·00	0·00	0·00
1	0·0	0·00	0·00	3·15	0·00
2	0·5	0·00	0·00	3·50	0·02
3	2·5	0·05	0·00	3·65	0·09
4	8·2	0·35	0·03	3·74	0·31
5	15·0	1·20	0·18	3·81	0·57
6	20·0	5·86	1·17	3·78	0·76
7	25·5	6·96	1·78	3·72	0·97
8	29·0	7·26	2·11	3·62	1·05
9	32·0	7·41	2·48	3·52	1·13
10	34·0	7·46	2·54	3·43	1·17
11	36·0	7·41	2·67	3·30	1·19
12	37·0	7·31	2·70	3·21	1·19
13	38·0	6·91	2·62	3·08	1·17
14	39·0	6·01	2·34	2·99	1·17
15		4·41	1·76	2·90	1·16
16		3·06	1·22	2·80	1·12
17		2·55	1·02	2·71	1·08
18		2·10	0·84	2·62	1·05
19	40·0	1·85	0·74	2·52	1·01
20		1·65	0·66	2·42	0·97
21 to 29		10·20	4·03	17·60	7·04
30 to 39		5·90	2·36	11·42	4·57
40 to 60		4·05	1·62	6·40	2·56
Total ..	—	99·96	34·92	99·89	31·35

[1] t, the percentage of subjects previously tonsillectomized, is derived from the "middle" smoothed curve in Figure II. The figures given for the age of twelve years onwards are got from some extrapolation in Figure II. The figure 40% given for age fifteen years and above is commented on later in the text.

[2] f, the percentage frequency for each age group, is taken from smoothed curves derived from data given in Table IX.

[3] ft is the product of f and t, expressed as a percentage, and represents the expected proportion of previously tonsillectomized subjects for each age group.

Using the data calculated in Table XXIV we find the following.

(i) With regard to bulbar paralysis in the 1947–1948 epidemic, the expected proportion of previously tonsillectomized subjects for this class is 34·92%. The observed ratio is 35 tonsillectomized out of 39 (90%). Testing these proportions for homogeneity, we find that $\chi^2 = 51 \cdot 5^{***}$ on one degree of freedom; $P < 0 \cdot 001$.

(ii) With regard to non-bulbar paralytic poliomyelitis in the 1947–1948 epidemic, the expected proportion of previously tonsillectomized subjects for this class is 31·35%. The observed ratio is 21 tonsillectomized out of 61 (34·4%). Testing these proportions for homogeneity, we find $\chi^2 = 0 \cdot 270$ on one degree of freedom, which is not significant.

It may therefore be concluded: (i) that there is a highly significant association between the contraction of bulbar poliomyelitis and previous tonsillectomy; (ii) that there is no evidence of any association between the contraction of non-bulbar paralytic poliomyelitis and previous tonsillectomy.

The finding (i) above accounts for the association shown between previous tonsillectomy and paralytic poliomyelitis not subdivided by clinical class.

Non-Paralytic Poliomyelitis.

It was decided, on account of the difficulties in diagnosis, among other reasons, not to prosecute further inquiries into the tonsillectomy status of the non-paralytic poliomyelitis patients. Nevertheless it is worth while examining the existing data. These are analysed in Table XXV. Reasons were given in the earlier study of this epidemic for believing that the 60 cases selected represented a fairly pure group of this form of the disease (see Southcott and Crosby, 1949).

If we ignore the very large class in "Not Known", it is seen that there were nine tonsillectomized subjects out of 25, and 16 not tonsillectomized. When we test the proportion of nine out of 25 against hypothetical proportions of, for example, 30% and 25%, it is found that χ^2 on one degree of freedom is respectively 0·429 and 1·613, neither of which values indicates a significant probability.

TABLE XXV.

Analysis of Cases of Non-Paralytic Poliomyelitis in the 1947–1948 Epidemic in South Australia, Divided into Tonsillectomized and Non-Tonsillectomized Subjects by Rural and Urban Distribution.

Rural or Urban.	Tonsillectomized Subjects.	Non-Tonsillectomized Subjects.	Not Known.	Total.
Rural	2	7	21	30
Urban	7	9	14	30
Totals ..	9	16	35	60

Thus there is no evidence, from the admittedly small number of figures available, of any association between the contraction of non-paralytic poliomyelitis and previous tonsillectomy. The data available are too meagre to show anything except an extremely large difference, if it were present.

TABLE XXVI.

Age Distribution of Paralytic Poliomyelitis in South Australia in 1947–1948, divided into Bulbar and Non-Bulbar, by Rural and Urban Distribution, and Tonsillectomized and Non-Tonsillectomized Subjects.

Rural or Urban.	Paralytic Poliomyelitis.	Subjects Tonsillectomized or Not.	Age (Years). 0 to 4	5 to 9	10 to 14	15 to 19	20 to 24	25 to 29	30 to 34	35 to 39	40 to 44	45 and Over.	Total.
Rural.	Bulbar ..	Tonsillectomized ..	0	3	10	1	1	1	0	1	—	—	17
		Not tonsillectomized	—	—	—	—	1	—	—	—	—	—	1
		Not known ..	—	1	—	—	—	—	—	—	—	—	1
		Total	0	4	10	1	2	1	—	1	—	—	19
	Non-bulbar..	Tonsillectomized ..	0	3	1	4[1]	3[1]	0	1	0	1	—	13
		Not tonsillectomized	1	3	2	3	1	2	1	1	0	1	15
		Not known ..	—	—	—	—	—	1	—	—	—	—	1
		Total	1	6	3	7	4	3	2	1	1	1	29
	All paralytic poliomyelitis	Total	1	10	13	8	6	4	2	2	1	1	48
Urban.	Bulbar ..	Tonsillectomized ..	0	9	4	3	0	0	1	0	0	0	17
		Not tonsillectomized	0	1	0	1	0	1	0	0	0	1	4
		Not known ..	—	—	—	—	—	—	—	—	—	—	0
		Total	0	10	4	4	0	1	1	0	0	1	21
	Non-bulbar..	Tonsillectomized	0	2	1	2	2	0	1	—	—	—	8
		Not tonsillectomized	10	4	3	1	1	2	3	0	0	1	25
		Not known ..	—	—	—	—	1	—	—	—	—	—	1
		Total	10	6	4	3	4	2	4	0	0	1	34
	All paralytic poliomyelitis	Total	10	16	8	7	4	3	5	0	0	2	55
Rural and Urban Combined.	Bulbar ..	Tonsillectomized ..	0	12	14	4	1	1	1	1	—	—	34
		Not tonsillectomized	0	1	0	1	1	1	0	0	0	1	5
		Not known ..	0	1	—	—	—	—	—	—	—	—	1
		Total	0	14	14	5	2	2	1	1	0	1	40
	Non-bulbar..	Tonsillectomized ..	0	5	2	6	5	0	2	0	1	—	21
		Not tonsillectomized	11	7	5	4	2	4	4	1	0	2	40
		Not known ..	—	—	—	—	1	1	—	—	—	—	2
		Total	11	12	7	10	8	5	6	1	1	2	63
	All paralytic poliomyelitis	Total	11	26	21	15[1]	10[1]	7	7	2	1	3	103

[1] The differences shown here from the table published in 1949 are due to correction of the age in one case on further information. The actual age was nineteen years eleven months instead of twenty years.

Age Incidence of Bulbar and Non-Bulbar Paralytic Poliomyelitis.

This analysis is required for a study of the duration of the association between bulbar poliomyelitis and previous tonsillectomy. It is proposed initially to compare the age distributions of the bulbar and non-bulbar paralytic cases of the 1947–1948 South Australian epidemics. Table XXVI shows the age distribution of these forms, divided by tonsillectomized and non-tonsillectomized, rural and urban.

Testing the difference of the age distributions between bulbar and non-bulbar paralytic poliomyelitis, we have, after necessary amalgamation of groups, the figures in Table XXVII.

TABLE XXVII.

Type of Poliomyelitis.	Age (Years).				Totals.
	0 to 9.	10 to 14.	15 to 19.	20 and Over.	
Bulbar	14	14	5	7	40
Non-bulbar paralytic ..	23	7	10	23	63
Total ..	37	21	15	30	103

Thus $\chi^2=10\cdot09$* on three degrees of freedom, and $P<0\cdot02$.

There is therefore a significantly different age distribution between the bulbar and non-bulbar paralytic cases. It will be noted from Table XXVI that none of the 35 tonsillectomized patients with bulbar paralysis was below the age of five years. In the age incidences of the nought to four and five to nine years age groups, we have the figures shown in Table XXVIII.

TABLE XXVIII.

Type of Paralysis.	Age (Years).		Total.
	0 to 4.	5 to 9.	
Bulbar	0	14	14
Non-bulbar	11	12	23
Total	11	26	37

Using an exact test we find that $P=0\cdot0022$, indicating a significant difference between the distributions in these classes. It can be shown that the modal age for tonsillectomy, both in controls without poliomyelitis and for bulbar poliomyelitis subjects, is about five or six years.

If we make a further comparison of the age distributions of bulbar with non-bulbar paralytic poliomyelitis in the age groups from ten years upwards, we have the findings in Table XXIX.

TABLE XXIX.

Type of Poliomyelitis.	Age (Years).		Total.
	10 to 14.	15 and Over.	
Bulbar	14	12	26
Non-bulbar paralytic	7	33	40
Total	21	45	66

Thus $\chi_c^2=7\cdot99$** on one degree of freedom, and $P<0\cdot01$.

This shows that there is a significant difference between the distributions even in these higher age groups. The difference in age distributions then between the bulbar and non-bulbar paralytic cases cannot be due simply to the exclusion in the former of those ages in which only a small percentage of subjects is tonsillectomized.

For the 1947–1948 epidemic paralytic cases we have the following statistics (Table XXX):

TABLE XXX.

Type of Paralysis.	Mean Age (m). (Years.)	Standard Deviation (s). (Years.)
Bulbar	14·67	1·4310
Non-bulbar paralytic ..	16·99	1·4929

It will be observed that the mean age for the bulbar paralysis patients is lower than that for the non-bulbar paralysis cases. Testing shows that the difference of the means is not significant.

Further Inquiries into Controls.

The preceding analyses have demonstrated an association between bulbar poliomyelitis and previous tonsillectomy. Figures for control scholars for the years 1947 and 1948 were not divided by age, nor were there data available on the interval since tonsillectomy. Further aid was sought from the Schools Medical Services of the Education Department of South Australia, and the Principal Medical Officer, Dr. W. Christie, kindly consented to a modification of the medical *questionnaire* form (form MB21) which the parents fill in for each child. To the previous question "Has your child had his/her tonsils removed?" a further was added, "In what year?" This alteration was made in June, 1952, and since that time some 9000 replies have been analysed by the writer, for rural and urban distribution, age, school, and interval since tonsillectomy. These data apply to 1952, between June and November; however, it is possible to calculate the data for other years by appropriate methods.

Data on Incidence of Previous Tonsillectomy in Controls.

The age of the scholar was calculated by subtracting the year of birth from 1952, and the age at tonsillectomy was calculated by subtracting the year in which tonsillectomy was performed from 1952. Thus a child born in 1944 and tonsillectomized in 1950 was considered as being aged eight years and as having undergone tonsillectomy at the age of six years. It will be seen that this method of calculation could introduce complications; but the advantage is one of simplicity.[1] Up to the present data on 9000 children have been analysed, and are shown in Tables XXXI and XXXII. Figure I illustrates these data graphically.

For comparison with the data on age distribution of bulbar poliomyelitis and previous tonsillectomy of the 1947–1948 epidemic, we require appropriate data on controls. These data on controls are derived from 1952 control data thus: the child considered earlier, born in 1944 and tonsillectomized in 1950, would have been aged three years in 1947 and non-tonsillectomized. By a consideration of each class in Tables XXXI and XXXII in turn, the data for 1947 shown in Tables XXXIII and XXXIV and in Figure II have been obtained. For this procedure to be valid, it is necessary for the death and migration rates to be small. The death rate is obviously low. The migration rate is indicated by forms MB21, which show the place of birth, and it is not considered that the rate is high enough to invalidate the procedure.

Table XXXV shows combined data on tonsillectomized controls for 1947, giving the age distribution of these and the ages at which tonsillectomy was performed. For this amalgamation of data to be valid statistically, it is necessary that the data to be amalgamated be tested for homogeneity between urban and rural classes. For reasons of space these tests are not shown fully here, but Table XXXVI shows an example of this testing. For the group of controls aged seven years the data on the intervals since tonsillectomy are distributed

[1] Depending on the dates during the year of birth and tonsillectomy, the age at tonsillectomy in the example given could range from just over five years to nearly seven years. However, the group would be centred around the age of six years.

TABLE XXXI.

Age Distribution and Age at Tonsillectomy for Education Department Control Scholars in 1952—Rural.

Data on Tonsillectomy.	Age (Years).														Total.
	5	6	7	8	9	10	11	12	13	14	15	16	17	18	
Subjects not tonsillectomized	227	640	490	481	384	324	310	252	170	143	49	25	11	0	3506
Subjects tonsillectomized	25	120	125	116	135	128	139	118	100	54	30	13	4	2	1109
Age at tonsillectomy (years):															
1	—	1	—	—	1	—	—	—	—	—	—	—	—	—	2
2	1	3	4	4	1	5	6	1	2	—	—	—	—	—	27
3	3	12	14	6	8	6	5	4	7	5	—	—	—	—	70
4	10	31	18	21	24	14	11	12	13	7	3	3	—	—	167
5	10	37	33	22	23	28	31	19	16	6	4	1	2	—	232
6	—	32	35	30	33	23	28	20	14	9	5	2	1	1	233
7	—	—	14	22	24	19	17	21	13	6	5	2	—	—	143
8	—	—	—	9	11	13	12	11	10	5	2	2	—	—	75
9	—	—	—	—	5	11	14	5	5	5	0	1	—	—	46
10	—	—	—	—	—	4	6	9	8	3	2	2	—	—	34
11	—	—	—	—	—	—	4	8	3	1	2	0	0	1	19
12	—	—	—	—	—	—	—	1	1	4	—	—	—	—	6
13	—	—	—	—	—	—	—	—	4	1	1	0	1	—	7
14	—	—	—	—	—	—	—	—	—	1	2	—	—	—	3
15	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1
Age at tonsillectomy not known	1	4	7	2	5	5	5	7	4	1	3	—	—	—	44
Total tonsillectomized and non-tonsillectomized subjects	252	760	615	597	519	452	449	370	270	197	79	38	15	2	4615
Percentage of previously tonsillectomized subjects	10·1	15·8	20·4	19·3	26·0	28·3	31·0	31·9	37·1	27·4	38·0		34·5		

between rural and urban classes, groups being amalgamated where necessary for the test (Table XXXVI). Here $\chi^2=8\cdot420$ on four degrees of freedom, which is not significant.

Testing each annual age group separately we have the figures shown in Table XXXVII. Here $\Sigma\chi^2=24\cdot78$ on 27 degrees of freedom, which is not significant.

For each annual age group tested above we have not noticed any change with the increasing interval since tonsillectomy of the proportion of rural to urban cases. The data are therefore homogeneous, and may be pooled as in Table XXXV. These data will be used as controls to the tonsillectomized subjects with bulbar poliomyelitis.

Duration of the Association between Bulbar Poliomyelitis and Previous Tonsillectomy.

It is proposed in this section to study the duration of the association between bulbar poliomyelitis and previous tonsillectomy which has been demonstrated. Table XXXVIII shows the age distribution of the bulbar paralysis subjects of the 1947–1948 epidemic, classified into tonsillectomized and non-tonsillectomized, and if in the former class, by age at tonsillectomy. In Table XXXVIII the ages shown are for completed years; this is slightly different from the method used in calculating these data for controls (Tables XXXI to XXXV). Table XXXIX gives data on the interval in months or years which elapsed between the time of tonsillectomy and

TABLE XXXII.

Age Distribution and Age at Tonsillectomy for Education Department Control Scholars in 1952—Urban.

Data on Tonsillectomy.	Age (Years).														Total.
	5	6	7	8	9	10	11	12	13	14	15	16	17	18	
Subjects not tonsillectomized	324	591	368	110	282	164	64	183	230	384	215	73	16	3	3007
Subjects tonsillectomized	44	104	122	42	176	103	46	134	143	305	144	60	7	0	1430
Age at tonsillectomy (years):															
1	—	—	—	1	—	—	—	—	1	1	—	1	—	—	4
2	—	4	4	—	6	2	4	4	5	9	4	2	—	—	44
3	12	10	15	3	22	11	5	14	10	21	4	4	1	—	132
4	19	27	12	11	36	9	5	25	15	32	21	11	—	—	223
5	8	33	35	4	46	20	8	25	20	45	20	8	2	—	274
6	—	16	28	12	27	26	5	16	32	50	21	3	1	—	237
7	—	—	18	8	17	11	7	24	16	40	16	7	—	—	164
8	—	—	—	0	11	9	2	5	9	33	13	5	2	—	89
9	—	—	—	—	2	9	3	6	11	17	8	2	—	—	58
10	—	—	—	—	—	4	2	4	9	16	12	6	—	—	53
11	—	—	—	—	—	—	2	3	5	10	7	2	—	—	29
12	—	—	—	—	—	—	—	1	4	3	6	1	—	—	15
13	—	—	—	—	—	—	—	—	1	5	1	3	—	—	10
14	—	—	—	—	—	—	—	—	—	4	1	1	—	—	6
15	—	—	—	—	—	—	—	—	—	—	3	—	—	—	3
Age at tonsillectomy not known	5	14	10	3	9	2	3	7	5	19	7	4	1	—	89
Total tonsillectomized and non-tonsillectomized subjects	368	695	490	152	458	267	110	317	373	689	359	133	23	3	4437
Percentage of previously tonsillectomized subjects	12·0	15·0	24·9	27·6	38·4	38·6	41·8	42·2	38·3	44·3	40·1		42·1		

TABLE XXXIII.

Data for Age and Previous Tonsillectomy for 1947, Calculated from 1952 Scholar Survey—Rural.

Data on Tonsillectomy.	Age (Years) in 1947.														Total.
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	
Subjects not tonsillectomized	252	759	611	587	484	397	365	288	192	153	56	25	12	0	3506
Subjects tonsillectomized	—	1	4	10	35	55	84	82	78	44	23	13	3	2	434
Age at tonsillectomy (years):															
1	—	1	0	0	1	0	0	0	0	0	0	0	0	0	2
2	—	—	4	4	1	5	6	1	2	0	0	0	0	0	23
3	—	—	—	6	8	6	5	4	7	5	0	0	0	0	41
4	—	—	—	—	24	14	11	12	13	7	3	3	0	0	87
5	—	—	—	—	—	28	31	19	16	6	4	1	2	0	107
6	—	—	—	—	—	—	28	20	14	9	5	2	1	1	80
7	—	—	—	—	—	—	—	21	13	6	5	2	0	0	47
8	—	—	—	—	—	—	—	—	10	5	2	2	0	0	19
9	—	—	—	—	—	—	—	—	—	5	0	1	0	0	6
10	—	—	—	—	—	—	—	—	—	—	2	2	0	0	4
11	—	—	—	—	—	—	—	—	—	—	—	0	0	1	1
12	—	—	—	—	—	—	—	—	—	—	—	—	0	0	0
13	—	—	—	—	—	—	—	—	—	—	—	—	—	0	0
Age at tonsillectomy not known	—	0	0	0	1	2	3	5	3	1	2	0	0	0	17
Total tonsillectomized and non-tonsillectomized subjects	252	760	615	597	519	452	449	370	270	197	79	38	15	2	4615
Percentage of previously tonsillectomized subjects	0	0·1	0·65	1·7	6·75	12·2	18·7	22·2	28·9	22·4	29·2		30·6		

TABLE XXXIV.

Data for Age and Previous Tonsillectomy for Controls for 1947, Calculated from 1952 Scholar Survey—Urban.

Data on Tonsillectomy.	Age in 1947 (Years).														Total.
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	
Subjects not tonsillectomized	368	695	486	148	391	224	81	203	261	425	234	78	16	3	3613
Subjects tonsillectomized	0	0	4	4	67	43	29	114	112	264	125	55	7	0	824
Age at tonsillectomy (years):															
1	—	0	0	1	0	0	0	0	1	1	0	1	0	0	4
2	—	—	4	0	6	2	4	4	5	9	4	2	0	0	40
3	—	—	—	3	22	11	5	14	10	21	4	4	1	0	95
4	—	—	—	—	36	9	5	25	15	32	21	11	0	0	154
5	—	—	—	—	—	20	8	25	20	45	20	8	2	0	148
6	—	—	—	—	—	—	5	16	32	50	21	3	1	0	128
7	—	—	—	—	—	—	—	24	16	40	16	7	0	0	103
8	—	—	—	—	—	—	—	—	9	33	13	5	2	0	62
9	—	—	—	—	—	—	—	—	—	17	8	2	0	0	27
10	—	—	—	—	—	—	—	—	—	—	12	6	0	0	18
11	—	—	—	—	—	—	—	—	—	—	—	2	0	0	2
12	—	—	—	—	—	—	—	—	—	—	—	—	0	0	0
13	—	—	—	—	—	—	—	—	—	—	—	—	—	0	0
Age at tonsillectomy not known	0	0	0	0	3	1	2	6	4	16	6	4	1	0	43
Total tonsillectomized and non-tonsillectomized subjects	368	695	490	152	458	267	110	317	373	689	359	133	23	3	4437
Percentage previously tonsillectomized	0	0	0·8	2·6	14·6	16·1	26·4	35·5	30·0	38·4	33·9		41·4		

TABLE XXXV.

Combined (Rural and Urban) Data for 1947 on Tonsillectomized Control Scholars, Divided by Age, and Age at Tonsillectomy.

Data on Tonsillectomy.	Age (Years) in 1947.														Total.
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	
Age at tonsillectomy (years):															
1	—	1	—	1	1	—	—	—	1	1	—	1	—	—	6
2	—	—	8	4	7	7	10	5	7	9	4	2	—	—	63
3	—	—	—	9	30	17	10	18	17	26	4	4	1	—	136
4	—	—	—	—	60	23	16	37	28	39	24	14	—	—	241
5	—	—	—	—	—	48	39	44	36	51	24	9	4	—	255
6	—	—	—	—	—	—	33	36	46	59	26	5	2	1	208
7	—	—	—	—	—	—	—	45	29	46	21	9	—	—	150
8	—	—	—	—	—	—	—	—	19	38	15	7	2	—	81
9	—	—	—	—	—	—	—	—	—	22	8	3	—	—	33
10	—	—	—	—	—	—	—	—	—	—	14	8	—	—	22
11	—	—	—	—	—	—	—	—	—	—	—	2	0	1	3
12	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0
13	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0
Age at tonsillectomy not known	—	0	0	0	4	3	5	11	7	17	8	4	1	0	60
Totals	0	1	8	14	102	98	113	196	190	308	148	68	10	2	1258

the onset of symptoms in the 35 tonsillectomized subjects with bulbar paralysis.

It will be observed that in only one of the 35 tonsillectomized subjects with bulbar paralysis was the interval less than one month (it was fifteen days), and in only three cases of the 35 was the interval less than six months.

TABLE XXXVI.

Rural or Urban.	Years Since Tonsillectomy.					Total.
	6+5+4	3	2	1	0	
Rural	5	12	19	20	21	77
Urban	18	25	25	16	24	108
Total	23	37	44	36	45	185

Testing the actual data on the intervals since tonsillectomy of the tonsillectomized bulbar class against the data for controls shown in Table XXXV we proceed to test each annual age group as shown in Table XL. The probability P of this or a more discrepant distribution is 0·2813, which is not significant.

At age six years, $P=1·0$, which is not significant (no bulbar cases).

The figures for the seven years age group are shown in Table XLI. Here $P=0·1560$, which is not significant.

At age eight years, $P=1·0$, which is not significant (no bulbar cases).

And so, similarly, amalgamating classes where necessary, we have the following figures: at nine years, $P=0·8147$; at age ten years, $P=0·5560$; at eleven years, $P=0·5744$.

TABLE XXXVII.

Age. (Years.)	χ^2_c	Degrees of Freedom.	Significance.
2	0	1	Not significant.
3	0	2	Not significant; exact $P=1·0$.
4	$\chi^2_c=1·366$	1	Not significant.
5	1·331	2	Not significant.
6	6·726	3	Not significant.
7	8·420	4	Not significant.
8	4·137	5	Not significant.
9	2·005	6	Not significant.
10	0·652	2	Not significant.
11	$\chi^2_c=0·145$	1	Not significant.

Thus, up to the age of eleven years we have observed no evidence of any change in the effect with the length of time since tonsillectomy; that is, there is no evidence of any diminution of the strength of the association between bulbar poliomyelitis and previous tonsillectomy up to the age of eleven years.

TABLE XXXVIII.

Bulbar Poliomyelitis in the 1947–1948 Epidemic in South Australia, arranged in Age Groups and whether Subjects were Tonsillectomized or Not. Tonsillectomized Cases Classified by Age at Tonsillectomy.

Data on Tonsillectomy.	Age (Years).																			Total.
	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21 to 24	25 to 29	30 to 34	35 and over	
Subjects not tonsillectomized	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	1	0	1	0	1	4
Subjects tonsillectomized	4	0	4	0	4	3	3	5	2	1	1	0	2	0	2	1	0	1	1	1	35
Age at tonsillectomy (years):																					
2	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
3	1	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	2
4	2	—	2	—	1	—	1	—	—	1	—	—	—	—	—	—	—	—	1	—	8
5	1	—	—	—	2	1	1	1	—	—	—	—	1	—	—	—	—	—	—	—	7
6	—	—	1	—	1	—	—	2	1	—	—	—	1	—	1	—	—	1	—	—	8
7	—	—	—	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	2
8	—	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	—	2
9	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0
10	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
11	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	1
12	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0
14	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0
15	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0
16	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0
17	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	2
23	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	1
Totals	4	0	4	0	5	3	3	5	2	1	1	0	2	0	2	2	0	2	1	2	39
	Scholars												Non-scholars								

TABLE XXXIX.

Data on the Interval between Tonsillectomy and the Onset of Bulbar Poliomyelitis, in 35 Cases, in the 1947–1948 South Australian Epidemic.

Rural or Urban.	Interval.											Totals.
	Months.				Years.							
	0 to 1	1 to 2	2 to 6	6 to 7	1 to 2	2 to 3	3 to 4	4 to 5	5 to 10	10 to 15	15 to 20	20 and Over.	
Rural	1	0	2	0	0	0	2	3	5	3	0	1	17
Urban	0	0	0	1	4	2	2	2	4	2	0	1	18
Totals	1	0	2	1	4	2	4	5	9	5	0	2	35

However, the classes with bulbar poliomyelitis tested above are small, and the fact that we have not observed any effect does not exclude the possibility that such an effect may be present.

Figure II shows smoothed curves drawn for the data on the proportions of subjects tonsillectomized by age distribution for 1947 controls. The data over the age of eleven years are unreliable, and proportions for classes over that age in 1947 need to be inferred by extrapolation. In making up Table XXIV the figure of 40% was chosen for the proportion of subjects tonsillectomized for ages twenty years and above. This proportion is probably a little high, but the use of this figure is a safe statistical procedure, as it tends to reduce significance in the results of the tests, and hence makes the conclusions more conservative. A survey of a series of hospital patients in 1952 and 1953 has given confirmation that this proportion is a safe one to adopt in these tests. It is proposed to use this figure of 40% as the expected proportion of the previously tonsillectomized in the following tests on the data of the bulbar poliomyelitis subjects of the age of twelve years and above.

TABLE XL.
Age Five Years.

	Years Since Tonsillectomy.		Total.
	4+3+2	1+0	
Controls	24	71	95
Bulbar poliomyelitis subjects ..	2	2	4
Total	26	73	99

Over the ages twelve to twenty years we have the following distribution of subjects of bulbar poliomyelitis in 1947–1948: tonsillectomized, 14; non-tonsillectomized, one; total, 15. Testing this distribution against a hypothetical expected proportion of tonsillectomized of 40%, we have the probability P of this or a more discrepant distribution $0{\cdot}4^{15}+15\,(0{\cdot}4)^{14}\,(0{\cdot}6)=0{\cdot}0000252$, which shows that the influence extends into this age group.

TABLE XLI.
Age Seven Years.

	Years Since Tonsillectomy.			Totals.
	6+5	4+3+2	1+0	
Controls	5	99	81	185
Bulbar poliomyelitis subjects ..	1	2	1	4
Total	6	101	82	189

Dividing the data given into various age groups for testing, we obtain the figures shown in Table XLII.

It can be seen from these tests that the effect is still significant in the group aged seventeen years and more, suggesting that the duration of the effect is likely to be prolonged, since the modal age for tonsillectomy is five or six years. However, as the intervals since tonsillectomy in the eight tonsillectomized members aged seventeen years and above range from one to twenty-seven years, conclusions cannot be drawn with certainty.

Stronger conclusions can be drawn from the class aged twelve to twenty years. In this class were 14 tonsillectomized subjects and one non-tonsillectomized subject. If we consider only those members with, for example, an interval since tonsillectomy of four or five years, we find 12 such members, who may be set against the one non-tonsillectomized member. Comparing the proportion 12 out of 13 against an expectation of 40% of tonsillectomized subjects, we find that $P=0{\cdot}000138$, a highly significant result, showing that the effect is still present in subjects subjected to tonsillectomy four or five years or more previously. Proceeding similarly with further groups with increasing interval since tonsillectomy in the twelve to twenty years age group, we find the results shown in Table XLIII.

We have thus found that in the age group twelve to twenty years the effect has lasted at least nine or ten years.[1] In the group aged less than twelve years the intervals since tonsillectomy are naturally shorter, and therefore one cannot expect to find evidence of a long duration of the effect. No patient who had had bulbar poliomyelitis was aged less than five years. In the bulbar cases in the age group five to eleven years, we did not notice any change in the effect with increasing interval since tonsillectomy. In the age group aged twenty-one years and over there are too few cases for conclusions to be drawn from testing. This may be an indication that the effect of tonsillectomy falls off, but it may also be a simple age effect (see below). No conclusion can be drawn as to the possible maximum duration of the association. Longer durations than have been shown here, even possibly life-long durations, have not been excluded and are possible.

TABLE XLII.

Age. (Years.)	Tonsillectomized Subjects.	Non-Tonsillectomized Subjects.	Total.	*P.*
12 to 20 ..	14	1	15	0·0000252
15 to 19 ..	5	0	5	0·01024
17 and over ..	8	3	11	0·0293
19 and over ..	6	3	9	0·0994
21 and over ..	3	2	5	0·3174

Age Distribution and Clinical Features of the 1947–1948 Cases of Bulbar Poliomyelitis without Previous Tonsillectomy.

The ages of the four non-tonsillectomized subjects of bulbar poliomyelitis were nine, twenty, twenty-eight and forty-five years. These figures suggest that there is an increased liability to contraction of bulbar poliomyelitis, with certain increases of age, separate from the influence exerted by previous tonsillectomy.

The clinical features of the four bulbar cases without previous tonsillectomy do not appear to differ much from those of the 35 bulbar cases with previous tonsillectomy, although it is difficult to draw firm conclusions from such a short series of the bulbar cases without previous tonsillectomy. Of the four cases without previous tonsillectomy, two were fatal. All four patients showed encephalitic or mid-brain signs as defined earlier; three showed spinal lesions. All four had palatal or pharyngeal palsy. Unfortunately neither of the two patients who died came to autopsy.

It is hoped to make further clinical analyses later with a larger series of bulbar cases without previous tonsillectomy.

TABLE XLIII.

Interval in Years.	Tonsillectomized Subjects.	Non-Tonsillectomized Subjects.	Total.	*P.*
4 or 5 and over ..	12	1	13	0·000138***
6 and over	10	1	11	0·000734***
7 and over	8	1	9	0·00380**
8 and over	7	1	8	0·00852**
9 or 10 and over ..	5	1	6	0·0410*
11 and over	3	1	4	0·179 (not significant)

[1] The fact that the probabilities increase with an increasing interval since tonsillectomy does not necessarily mean that the strength of the association is diminishing, but may be solely due to the fact that we have tested against a constant proportion of 0·4, although as the number of years increases, the proportion

$$\frac{\text{tonsillectomized } x \text{ years ago or more}}{\text{tonsillectomized } x \text{ years ago or more} + \text{non-tonsillectomized}}$$

decreases. Furthermore, there is in the tests performed a decreasing sensitivity with decreasing class numbers.

TABLE XLIV.

Transmissions between Cases in the 1947–1948 South Australian Poliomyelitis Epidemic, the Poliomyelitis Cases being Divided by Clinical Type.

From.	To.		From.	To.		Total.
		(*a*) Transmissions Involving Bulbar Poliomyelitis.				
Non-paralytic ..	Bulbar	4	Bulbar	Non-paralytic	1	5
Non-bulbar paralytic	Bulbar	2	Bulbar	Non-bulbar paralytic	1	3
Bulbar	Bulbar	0				0
		(*b*) Transmissions Not Involving Bulbar Poliomyelitis.				
Non-paralytic ..	Non-bulbar paralytic	5	Non-bulbar paralytic	Non-paralytic	2	7
Non-bulbar paralytic	Non-bulbar paralytic	1				1
Non-paralytic ..	Non-paralytic	9				9
Total transmissions						25

SOME EPIDEMIOLOGICAL ASPECTS OF BULBAR POLIOMYELITIS.

The present study has shown that in the 1947–1948 South Australian poliomyelitis epidemic the tonsillectomized class was predisposed to the contraction of bulbar poliomyelitis. The high incidence of bulbar paralysis among the paralytic class made this effect noticeable in the figures for the whole of the paralytic series. Top and Vaughan (1941) showed that in the Detroit (United States of America) epidemic of 1939 the incidence of previous tonsillectomy in poliomyelitis cases was greater than in various controls selected. Top (1952) has demonstrated an association between bulbar poliomyelitis and previous tonsillectomy. The American and South Australian figures for the proportion of bulbar cases with previous tonsillectomy are similar. Top uses a somewhat different clinical classification from that adopted here, his cases with bulbar manifestations being divided into bulbar or spino-bulbar. In the present study all cases with any bulbar manifestation have been included in "bulbar poliomyelitis". Top gives the figure of 85·1% with previous tonsillectomy for his bulbar cases and 68·7% for his spino-bulbar cases. In the South Australian cases the figure was 89·7%. Top was not able to give an estimate of the duration of the association between previous tonsillectomy and the contraction of bulbar poliomyelitis, because he had not data of previous tonsillectomy for controls. However, he does list his data on intervals, which give the impression of being roughly comparable with the South Australian data.

The clinical features and the age distribution of the bulbar poliomyelitis cases are so different from those of non-bulbar paralytic poliomyelitis that one may ask whether it is possible that human bulbar poliomyelitis is a different disease, occurring in conjunction with "normal" poliomyelitis during an epidemic—as, for example, the Coxsackie virus infections occur.

In the epidemiological study of the 1947–1948 South Australian epidemic, the writer and N. D. Crosby were of the opinion that the epidemic was spread by contact (droplet), a total of 25 "observed" transmissions being recorded. A review of these by the present writer has shown bulbar cases arising from, or giving rise to, non-bulbar cases, both paralytic and non-paralytic. The known transmissions are divided by clinical type in Table XLIV.

In Table XLIV it will be seen that there was not an undue proportion of transmissions recorded as giving rise to bulbar cases, as would be expected if only bulbar poliomyelitis originated from contact transmission. It will be noted also that there was no bulbar-to-bulbar transmission recorded. Were the suggestion that human bulbar poliomyelitis constitutes a separate disease true, one would expect this to be the only type of transmission involving bulbar poliomyelitis to be recorded. The evidence recorded in this table then gives no support to the suggestion that the bulbar cases are due to a separate virus, rather the contrary. This is in agreement with experimental studies in animals, in which one finds that inoculation with the same virus may give rise to both bulbar and non-bulbar forms of poliomyelitis.

One further question may be asked: "Is the age distribution of the bulbar cases such as one would expect from a consideration of the age-specific attack rate of non-bulbar paralytic poliomyelitis in the tonsillectomized proportion of each age group?" The answer to this question is to be found in Table XXIV, in which column F could represent the distribution of bulbar cases expected from such an hypothesis. The distribution of proportions shown in column F is obviously very different from the observed distribution of proportions of the bulbar cases by age shown in column C, or the distribution of the expected proportions of the bulbar cases with previous tonsillectomy represented in column D. There is a much more pronounced "peak" in the bulbar distribution than that shown in column F. There appears no simple method by which one distribution can be derived from the other.

Portal of Entry of the Virus.

The writer is of the opinion that the most satisfactory explanation of the portal of entry of the virus in the bulbar cases is that the virus has gained entry to the medulla via the motor pathway from the tonsillar area of the pharynx to the *nucleus ambiguus*.

The work of Faber and Silverberg (1946) on the possible portals of entry of the virus in human poliomyelitis may be regarded as authoritative. These authors envisage the whole length of the alimentary canal, from the olfactory area to the rectum, as possible portals of entry. They studied the histopathological features of eight fatal cases (five bulbar, three spinal) and recorded that the trigeminal afferent system (nose, mouth and pharynx) was frequently involved, and also the visceral afferent system of the ninth and tenth cranial nerves (nose, mouth, pharynx, œsophagus, bronchi, stomach and intestines) was fairly commonly involved; the gustatory system (seventh, ninth and tenth cranial nerves) was occasionally involved. The vagal efferent or parasympathetic system and the olfactory system were not involved. These authors considered that the evidence of penetration through the upper part of the alimentary tract was more obvious than that of penetration through the lower part of the alimentary tract. The pharynx appeared to be the specially favourable site for the primary invasion of the virus. It was not thought probable that nerves without superficial connexion are likely to transmit the virus, with the exception of the motor nerves in the pharynx after (recent) tonsillectomy.

Fowler (1951) studied a series of 10 fatal cases from the 1947–1948 South Australian epidemic. Fowler gives sufficient clinical detail in these cases for the writer to recognize each of them. By the standards adopted in the present article, in eight of the cases the paralysis was bulbar and in two it was spinal. Fowler remarks that many of his patients died early in the paralytic stage. In all except one of his cases there were only moderately severe lesions or no lesions at all on the afferent side of the spinal and main sensory trigeminal nuclei, although there was frequently severe damage to the trigeminal motor nuclei. Fowler declares, in relation to Faber and Silverberg's (1946) belief that the trigeminal route of invasion of the virus into the central nervous system is the most important, that deductions from peripheral ganglia are unreliable, as lesions occur at various levels of the peripheral ganglia, and furthermore, that the photographs shown by those authors of severe lesions in ganglia are "most unconvincing", appearing to him to be more typical of very mild lesions. Bodian and Howe (1947) are similarly dubious about how much can be ascribed to ganglionic lesions, and suggest that these are caused

by centrifugal rather than centripetal spread of the virus. Elsewhere in his paper Fowler describes the heavy lesions in the *nucleus ambiguus* in the South Australian cases. Lesions of the nucleus of the *tractus solitarius* he describes as "usually of rather slight degree". Fowler concludes that "evidence concerning the portal of entry in the bulbar cases is very suggestive of passage of virus from the upper part of the alimentary tract, most likely from the pharynx via the fibres entering the nucleus of the *tractus solitarius*, or less likely, but not excluded, via the motor nerves direct to the *nucleus ambiguus*".

It is noteworthy that these earlier suggestions of the virus passing along the motor pathway to the medulla were made after a study of the histopathological features of human fatal cases, which are predominantly bulbar. However, the final conclusions were always that the sensory pathways (including gustatory and splanchnic) were the more likely for the entry of the virus to the central nervous system. The belief that the entry of virus to the central nervous system is via nervous pathways rests solidly on a great deal of experimental and other evidence.

Poliomyelitis following recent tonsillectomy appears to be easy to explain by the local inoculation of virus preexisting in the pharynx. It is more difficult to explain the long-range association between bulbar poliomyelitis and previous tonsillectomy which has been shown. The most obvious (but I believe incorrect) explanation is that the tonsils constitute a specific barrier against infection with poliomyelitis which has been removed. I do not know of any other evidence which shows that tonsillectomy leaves a person more susceptible to the contraction of some infectious disease. On the other hand, that the retention of tonsils is associated with the liability to disease has been shown, for example by Stewart and Hewitt (1952). These authors have demonstrated that a child who has suffered from rheumatic fever with previous tonsillitis is more prone to recurrences of rheumatism if not subjected to tonsillectomy.

It is reasonable to conclude from the evidence of the association between bulbar poliomyelitis and previous tonsillectomy that in the majority of the bulbar cases the virus has gained entry via the tonsillar area of the pharynx. To cause the medullary lesions the virus could conceivably travel via either the sensory or the motor nerves. Some previous viewpoints on this have been introduced. Howe and Bodian (1942) have suggested that poliomyelitis virus is transmitted equally well by motor and sensory fibres.

The sensory nerve supply of the tonsillar area is from the tonsillar branch of the glossopharyngeal nerve. This tonsillar branch forms a plexus to supply the mucous membrane covering the tonsil, the adjacent part of the soft palate, and the palatine arches. The afferent pathway is via the inferior and superior glossopharyngeal ganglia to the *nucleus dorsalis*, although some fibres (chiefly for taste) join the *tractus solitarius*, to terminate in the nucleus of the *tractus solitarius*.

The tonsil receives decussating muscular fibres from the palatopharyngeus and the palatoglossus, which enter the lower third of its capsule. These fibres may be only few, or may be more numerous. These muscles are innervated by the vagus nerve, the neurons originating in the *nucleus ambiguus*, in either its vagal or its accessory part. The *nucleus ambiguus* gives origin to the branchial motor fibres of the glossopharyngeal and vagus nerves. The only motor distribution from the glossopharyngeal nerve is to the stylopharyngeus muscle, which does not concern us here.

Fowler (1951), in agreement with other writers, has pointed out that in the bulbar cases there is rarely heavy damage to the dorsal nucleus of the glossopharyngeal and vagus, and even occasionally there is no lesion at all to be seen there. There is a little evidence that poliomyelitis virus may be present in parts of the central nervous system without causing a destructive lesion.

The process by which the motor neurons of the tonsillar region are rendered more accessible to entry by the virus is considered by the writer to be as follows. During tonsillectomy the muscular attachments to the capsule of the tonsil are damaged, and after healing these (and possibly other neighbouring pharyngeal) muscular fibres remain much closer to the surface than prior to the operation, along with the motor neurons supplying them. After gaining entry to the motor axons, the virus passes to the *nucleus ambiguus*, from which it may then spread to the neighbouring reticular nuclei and elsewhere in the central nervous system. It is not possible at present to suggest at what point the virus gains entry to the motor axon.

This hypothesis of entry of virus to the medulla along the motor pathway explains the heavy damage to the *nucleus ambiguus* and the distribution of the other medullary lesions discussed earlier. It explains the influence of tonsillectomy, both recent and non-recent, in predisposing the subject to bulbar poliomyelitis. Furthermore, the hypothesis rationalizes many of the experimental findings. Thus Sabin (1938) observed a high percentage of bulbar paralysis in monkeys after pharyngeal inoculation of the virus. Von Magnus and Melnick (1948) observed that in their experiments only two monkeys out of 14 fed with poliomyelitis virus immediately after tonsillectomy contracted bulbar poliomyelitis. In Sabin's experiments the virus can be considered as having been given access to muscle. In von Magnus and Melnick's experiments virus was applied only to the surface of the pharynx, and it can be considered that the virus was not given good access to muscle. In the experiments of Faber *et alii* (1951) the virus was applied to the surface of the pharynx immediately before tonsillectomy, and this was followed by the invariable contraction of bulbar poliomyelitis. Here there was every opportunity for the virus on the pharyngeal surface to be introduced into muscle by the operative trauma. The hypothesis of motor transmission of the virus after access had been given to muscle fibres explains all these experimental findings without difficulty. An hypothesis of transmission of virus along sensory fibres offers no such easy explanation for the differences in the experimental findings.

Bulbar poliomyelitis is occasionally seen in the experimental animal after the application of the virus to the intact pharyngeal epithelium. In human cases of bulbar poliomyelitis with prior (non-recent) tonsillectomy the epithelial surface of the tonsillar area has been reconstituted. To accept the hypothesis that in bulbar poliomyelitis the virus enters the medulla along the pharyngeal motor nerve fibres, we must also accept that virus can pass through an intact epithelial surface to the subjacent motor neurons. Little appears to be known about such transepithelial passage of poliomyelitis virus. Whether also the epithelium of the tonsillar area after tonsillectomy is less resistant to virus entry is not known.

Whether the variability of the extent of the muscular attachment to the capsule of the tonsil influences the susceptibility of the tonsillectomized to bulbar poliomyelitis is not known; possibly also the degree of exposure of pharyngeal musculature at tonsillectomy, and the anatomical relationships in the post-tonsillectomy pharynx—for example, depth of tissue between epithelium and muscle—may exert some influence.

The hypothesis of virus entry to the medulla along the motor pathway accounts for only the bulbar cases. Most discussions on the portal of entry in human poliomyelitis do not distinguish very sharply between bulbar and non-bulbar cases. Various possible pathways from the oropharynx to the central nervous system have been suggested in the past. Thus Toomey (1936) suggested that the virus gains entry via the *chorda tympani* branch of the facial nerve, although this hypothesis does not appear to have gained much favour.

One of the pathways considered at the present time to be a likely pathway of virus to the central nervous system is the mesencephalic tract of the trigeminal nerve. It is believed that this is made up of sensory fibres derived from the dental and palatine nerves ("Cunningham's Anatomy"). Advocates of the theory that this is the pathway point out that it offers advantages for the entry of the virus to the central nervous system, in that it constitutes a direct route, free of synapses. A synapse is considered as offering a barrier to the path of the virus (Faber and Silverberg, 1946). The neuronal endings of this tract are not superficial. The pathway from the motor neuron of the pharynx to the *nucleus ambiguus* also offers a direct route, free of synapses.

Alternative hypotheses as to the role of tonsillectomy in predisposing the subject to bulbar poliomyelitis can be formulated. Thus it could be postulated that tonsillectomy facilitates the entry of virus in some way to the sensory fibres. Or again one could postulate that a person has a general susceptibility to pharyngeal infection, leading on the one hand to tonsillitis and hence frequently to tonsillectomy, and on the other hand to bulbar poliomyelitis. However, neither of these

alternative hypotheses appears to explain much, and neither need be seriously considered.

It should not be difficult to devise an experiment to test in the previously tonsillectomized monkey whether it is easier to contract bulbar poliomyelitis with the appropriate motor or sensory pathway destroyed, following the swabbing or spraying of the tonsillar area with virus; such an experiment could be unilateral. An alternative method, not beyond the bounds of present possibility, would be the actual tracing along nerves of poliomyelitis virus "labelled" by some radioactive element.

Howe and Bodian (1942) have shown that the rate of travel of poliomyelitis virus along the axon of a nerve fibre is about 2·4 millimetres per hour. This, in the experimental animal, was preceded by a latent period of about eleven hours before significant movement of virus commenced from the inoculated cut nerve ending. Taking the length of the motor neuron from the tonsillar region to the medulla as about seven centimetres, we can then account for less than two days, whereas the incubation period in the bulbar cases is about ten to twelve days. We have therefore about eight to ten days to account for, which presumably is spent by the virus in the passage across the pharyngeal epithelium and in proliferation prior to entry into the motor axon, or in proliferation and spread within the medulla before symptoms become manifest. Again, it should not be difficult to devise an experiment to find what period elapses before movement of virus along the nervous pathway to the medulla commences.

Other Possibilities of Motor Neuron Entry of Poliomyelitis Virus to the Central Nervous System.

It is interesting to note the findings of various writers—for example, Martin (1950), McCloskey (1950, 1951), Hill and Knowelden (1950) and others—on prophylactic and other injections predisposing to the contraction of paralytic poliomyelitis. It is an interesting speculation that in the cases of paralytic poliomyelitis following injections, circulating virus may have been given access to the motor neuron by the trauma of the injection. Martin (1950) stated that the association was between poliomyelitis and intramuscular injections, although he did not elaborate further on the intramuscular aspect of the injection. It is not difficult to conceive that in, for example, prophylactic inoculation, actual trauma (either physical or chemical) can occur in or near muscle. A preceding viræmia would be necessary in these cases to account for these phenomena, there being no evidence that the virus is introduced along with the inoculum.

If this suggestion of entry of poliomyelitis virus along motor neurons to the central nervous system after injections is correct, then it may be of considerable importance, for example in public health work, in regard to the site and depth of the inoculations used.

A Note on the 1949–1950 South Australian Poliomyelitis Cases.

Cases of poliomyelitis continued to occur in considerable number in South Australia in 1949 and subsequently. Through the kindness of Dr. H. W. Linn, Medical Superintendent, Northfield Wards, Royal Adelaide Hospital, I have been permitted access to the records of the bulbar cases of 1949 and 1950. The same circular *questionnaire* with regard to previous tonsillectomy *et cetera* was sent out to these patients as was sent out to the 1947–1948 patients. So far answers have been received for 19 of the 30 patients who had bulbar paralysis, and of these 19 patients, 14 had previously been tonsillectomized and five had not. This proportion of 14 out of 19, or 74%, is in agreement with the earlier findings. In only one of these cases was the tonsillectomy recent (eleven days prior to the onset of symptoms).

Summary.

1. A survey of the 1947–1948 South Australian poliomyelitis cases has shown that of the 40 patients with bulbar paralysis 35 had previously undergone tonsillectomy, and four had not, and for one patient this information was not obtained. This proportion of 35 out of 39, or 90%, is significantly higher than expectation. Of the 35 patients only one had been tonsillectomized recently (fifteen days' interval between tonsillectomy and onset of poliomyelitis). In the remainder of the 35 cases there were longer intervals since tonsillectomy; thus 16 of the 35 tonsillectomized patients with bulbar paralysis had contracted the disease five or more years since tonsillectomy.

2. A history of previous knowledge of the association between tonsillectomy and poliomyelitis is presented.

3. A survey of data for 9000 school children has been made in South Australia to obtain control data of the proportion tonsillectomized in each annual age group. These data, presented in this paper, are used for comparisons to derive the duration of the association between bulbar poliomyelitis and previous tonsillectomy. Proportions of the tonsillectomized are higher for urban school children than for rural.

4. In South Australia for 1947–1948 the proportion of bulbar cases was high—40 cases out of a total of 103 paralytic cases (all forms). Bulbar poliomyelitis was responsible for most of the poliomyelitis deaths. The high proportion of bulbar cases made the effect of tonsillectomy noticeable over the figures for the total paralytic series for the epidemic.

5. No evidence has been found of an association between previous tonsillectomy and poliomyelitis other than bulbar; both paralytic (non-bulbar) and non-paralytic figures have been tested.

6. The age distributions of bulbar and non-bulbar paralytic poliomyelitis series are compared and are shown to differ significantly. No bulbar paralysis patient was aged less than five years. The curve for the age distribution of the bulbar cases is much more peaked than that of the non-bulbar paralytic cases.

7. Studies on the duration of the association between bulbar poliomyelitis and previous tonsillectomy are presented. In the lower age groups only short intervals since tonsillectomy are seen. It is shown that the effect of tonsillectomy is present in the age group of bulbar paralysis patients aged seventeen years and more, and as the modal age for tonsillectomy is five or six years this suggests a long duration of the effect. In the group of bulbar paralysis patients aged twenty-one years and over, the figures are too small for firm conclusions to be drawn. The figures are tested in the age group twelve to twenty years, and it is shown that in this group the effect of tonsillectomy lasts at least nine or ten years. Beyond that duration the classes have too few numbers for firm conclusions to be drawn, but even longer durations for the association, even possibly lifelong durations, are not excluded and are possible.

8. The age distribution of the non-tonsillectomized bulbar paralysis subjects suggests the possibility of an age effect of increasing liability to contraction of bulbar poliomyelitis with certain increases of age, independent of tonsillectomy. The clinical features of the non-tonsillectomized bulbar paralysis subjects are briefly discussed.

9. Transmissions between cases of poliomyelitis were recorded earlier. These are here classified by clinical type. Bulbar cases may arise from, or give rise to, cases of other clinical type. There appears no particular tendency for bulbar cases to originate other bulbar cases rather than other clinical types of poliomyelitis; in fact, no case of bulbar-to-bulbar transmission was recorded in the 25 transmissions "observed".

10. It is considered that the association between bulbar poliomyelitis and previous tonsillectomy is in favour of pharyngeal entry of the virus in the bulbar cases. The viewpoint is put forward that the virus gains entry to the medulla in the bulbar cases via the motor nerve supply of the tonsillar area of the pharynx to the *nucleus ambiguus*. The mechanism of this is discussed. It is considered that there is no evidence that the tonsils are specifically protective against entry of poliomyelitis virus, but that the trauma of tonsillectomy renders the motor nerves of the tonsillar region more accessible to the entry of virus to them from the pharyngeal surface. Previous findings with regard to both human histopathological studies and experimental studies with animals are discussed, and it is shown that this hypothesis of entry of virus via the motor innervation to the *nucleus ambiguus* explains many of the observed phenomena. It is conceived that the virus can pass through the pharyngeal mucosa of the tonsillectomized subject to reach the motor nerves, although the precise manner in which the motor neuron is reached is uncertain.

11. It is suggested that in poliomyelitis following prophylactic and other injections the virus also travels to the central nervous system along motor nerve fibres.

12. A brief survey of the 1949–1950 South Australian bulbar poliomyelitis cases confirms the high incidence of previous tonsillectomy in these cases.

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REFERENCES.

ANDERSON, J. A. (1945), "Poliomyelitis and Recent Tonsillectomy", *J. Pediat.*, 27 : 68.

AYCOCK, W. L. (1942), "Tonsillectomy and Poliomyelitis. I. Epidemiological Considerations", *Medicine*, 21 : 65.

AYCOCK, W. L., and LUTHER, E. H. (1929), "The Occurrence of Poliomyelitis following Tonsillectomy", *New England J. Med.*, 200 : 164.

AYER, W. D. (1929), "Poliomyelitis", "Inter-State Post-Graduate Medical Association of North America, Proceedings of the International Assembly (1928)", 319.

BAKER, A. B. (1949), "Bulbar Poliomyelitis. Its Mechanism and Treatment", *Am. J. Med.*, 6 : 614.

BAKER, A. B., MATZKE, H. A., and BROWN, J. R. (1950), "Poliomyelitis. III. Bulbar Poliomyelitis, a Study of Medullary Function", *Arch. Neurol. & Psychiat.*, 63 : 257.

BODIAN, D., and HOWE, H. A. (1947), "The Significance of Lesions in Peripheral Ganglia in Chimpanzee and in Human Poliomyelitis", *J. Exper. Med.*, 85 : 231.

CUNNING, D. S. (1947), "Tonsillectomy and Poliomyelitis", *Arch. Otolaryng.*, 46 : 575

"CUNNINGHAM'S ANATOMY", Eighth Edition, 901.

FABER, H. K. (1949), "Adenotonsillectomy and Poliomyelitis" (Editorial), *Pediatrics*, 3 : 256.

FABER, H. K., MCNAUGHT, R. C., SILVERBERG, R. J., and DONG, L. (1951), "Experimental Production of Post-tonsillectomy Bulbar Poliomyelitis", *Proc. Soc. Exper. Biol. & Med.*, 77 : 532.

FABER, H. K., and SILVERBERG, R. J. (1946), "A Neuropathological Study of Acute Human Poliomyelitis, with Special Reference to the Initial Lesions and to Various Potential Portals of Entry", *J. Exper. Med.*, 83 : 329.

FOWLER, M. (1951), "A Histopathological Study of Eleven Cases of Poliomyelitis, Including One in the Newborn", M. J. AUSTRALIA, 2 : 613.

FRANCIS, T., KRILL, C. E., TOOMEY, J. A., and MACK, W. N. (1942), "Poliomyelitis Following Tonsillectomy in Five Members of a Family", *J.A.M.A.*, 119 : 1392.

GLOVER, J. A. (1948), "Poliomyelitis and Tonsillectomy", *Month. Bull. Min. Health & Pub. Health Lab. Serv.*, 7 : 150.

HILL, A. B., and KNOWELDEN, J. (1950), "Inoculation and Poliomyelitis: A Statistical Investigation in England and Wales in 1949", *Brit. M. J.*, 2 : 1.

HOWE, H. A., and BODIAN, D. (1942), "Neural Mechanisms in Poliomyelitis", New York, The Commonwealth Fund.

Lancet, The (Editorial) (1949), "Poliomyelitis and Tonsillectomy", 2 : 424.

MARTIN, J. K. (1950), "Local Paralysis in Children after Injections", *Arch. Dis. Childhood*, 25 : 1.

MCCLOSKEY, B. P. (1950), "The Relation of Prophylactic Inoculations to the Onset of Poliomyelitis", *Lancet*, 1 : 659.

MCCLOSKEY, B. P. (1951), "The Relation of Prophylactic Inoculations to the Onset of Poliomyelitis: A Study of 620 Cases in the Victorian Epidemic of Poliomyelitis in 1949", M. J. AUSTRALIA, 1 : 613.

MILLER, A. H. (1951), "Tonsillectomy and Adenoidectomy and Poliomyelitis", *Arch. Otolaryng.*, 53 : 160.

POWELL, M. L. (1938), "The Diagnosis of Poliomyelitis", M. J. AUSTRALIA, 2 : 276.

SABIN, A. B. (1938), "Experimental Poliomyelitis by the Tonsillopharyngeal Route, with Special Reference to the Influence of Tonsillectomy on the Development of Bulbar Poliomyelitis", *J.A.M.A.*, 111 : 605.

SHEPPARD, P. A. E. (1912), "A Study of an Epidemic of Infantile Paralysis (Acute Anterior Poliomyelitis) in Springfield, Mass., in 1910", Massachusetts State Board of Health, Boston.

SIEGEL, M., GREENBERG, M., and MAGEE, M. C. (1951*a*), "Tonsillectomy and Poliomyelitis. I. Studies on Incidence in 1949", *J. Pediat.*, 38 : 537.

SIEGEL, M., GREENBERG, M., and MAGEE, M. C. (1951*b*), "Tonsillectomy and Poliomyelitis. II. Frequency of Bulbar Paralysis, 1944–1949", *J. Pediat.*, 38 : 548.

SOUTHCOTT, R. V. (1952), "Some Epidemiological and Public Health Aspects of Poliomyelitis", M. J. AUSTRALIA, 2 : 482.

SOUTHCOTT, R. V., and CROSBY, N. D. (1949), "Studies on the Epidemiology of the 1947–1948 Epidemic of Poliomyelitis in South Australia", M. J. AUSTRALIA, 2 : 481.

STEWART, A., and HEWITT, D. (1952), "Sore Throat and Rheumatic Fever", *Lancet*, 2 : 143.

TOOMEY, J. A. (1936), "The Seventh Nerve as a Possible Pathway for the Transmission of the Virus of Poliomyelitis", *Am. J. Dis. Child.*, 51 : 58.

TOP, F. H. (1952), "Occurrence of Poliomyelitis in Relation to Tonsillectomies at Various Intervals", *J.A.M.A.*, 150 : 534.

TOP, F. H., and VAUGHAN, H. F. (1941), "Epidemiology of Poliomyelitis in Detroit in 1939", *Am. J. Pub. Health*, 31 : 777.

VAN ROOYEN, C. E., and RHODES, A. J. (1948), "Virus Diseases of Man", New York, Second Edition.

VON MAGNUS, H., and MELNICK, J. L. (1948), "Tonsillectomy in Experimental Poliomyelitis", *Am. J. Hyg.*, 48 : 113.

WICKMAN, I. (1907), "*Beiträge zur Kenntnis der Heine-Medinschen Krankheit*", Karger, Berlin.

WICKMAN, I. (1913), "Acute Poliomyelitis", Translated by W. J. M. A. Maloney, Nervous and Mental Diseases Monograph Series, Number 16, New York.

WILSON, J. L. (1952), "Relationship of Tonsillectomy to Incidence of Poliomyelitis", *J.A.M.A.*, 150 : 539.

A BLOODLESS OPERATION FOR PARONYCHIA.

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PARONYCHIA is a common condition which is usually treated by cutting operations, of which Kanavel's is one. These operations often cause unnecessary suffering, loss of time and expense for the patient. The cutting operation is inartistic, in that the bead of pus which is evacuated is insignificant when compared with the ritual and the incision for its evacuation. The sledge hammer is used to crack the peanut. An incision to evacuate pus should usually be made where the pus is nearest to the surface. Incisions through the external surface of the eponychium violate this principle, and are clumsy because they traverse a thick, vascular, sensitive and inflamed fold of skin, whereas they need only traverse a thin, avascular, insensitive layer of epithelium if made via the groove between the nail and the eponychium. The relationship of these structures and methods is illustrated in Figure I.

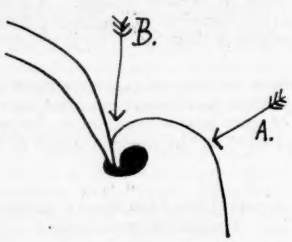

FIGURE I.

Transverse section of edge of nail and eponychium. The shaded area is pus, partly under eponychium, partly under nail edge. Arrow "A" is the approach by cutting (of Kanavel type). Arrow "B" is the bloodless approach.

The Method.

Anæsthesia and tourniquet are not used.

The eponychium is pushed back gently from the surface of the nail, in the direction of the centre of the swelling. I have used the tip of a closed pair of artery forceps for this. The eye-end of a large straight skin needle is then passed under the eponychium in the direction of the centre of the swelling. The œdema and softening tend to open the groove between the nail and the eponychium and make the passage easy. The eye-end of the needle is then levered outwards slightly two or three times until it breaks through the epithelium (the only layer which here covers the pus) and the operator is rewarded by seeing the bead of pus well up into the groove. During the following day or two, if discharge crusts in the groove, it is removed by

the patient, who uses the blunt end of a needle. This is all that is needed to effect a cure in most cases.

If the pus has spread under the nail, this fact can be recognized because the end of the needle, used as a probe, feels that the edge of the nail is free, and the needle-end can be passed painlessly under the edge, because the nail edge has already been separated from its bed by the pus. The paronychia will heal after this operation if the pus has spread only a little way under the nail, but not if the pus has extended further. The edge of the nail must then be removed. This can be done, still bloodlessly and without anæsthesia, by a strong, curved, pointed manicure scissors. The convexity of the curve of the blades is held towards the edge of the nail. One blade is passed easily under the edge of the nail into the space already made by the pus, and the other is slid down between the eponychium and the nail, also being passed easily into a space made by the pus. The edge of the nail is then cut away.

Results.

I found, during years of general practice, that the great majority of patients with paronychia could be cured by this minor procedure. In the more advanced cases, in which the pus has spread under the nail from one edge to the other, an operation of Kanavel type may be needed—namely, cutting of the eponychium to expose and remove some nail.

A NOTE ON THE POSSIBILITY OF OBTAINING FALSE BLOOD GROUP RESULTS FOR BABIES BY TESTING THE CORD BLOOD.

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As a routine procedure in these laboratories, any patient whose blood is found to be Rh-negative in the ante-natal clinic is checked at delivery for blood group and Rh type, and the placental blood is also tested to determine the baby's group and Rh type. On several occasions the proportion of negative results on specimens of placental blood seemed greater than would be expected, and a query was raised whether any mixing of maternal and placental blood occurred before the placental blood was taken, which could mask the true placental blood type, often weak in reaction. The placental blood is collected from the baby's end of the cord, after it has been cut, when the pulse can no longer be felt. As the placenta may be coming away from the uterine wall by this time, the damaging of placental and uterine tissues could cause intermingling of the respective blood supplies, with the resultant possibility that some maternal blood may be present in the cord vessels.

An analysis of all specimens of maternal blood (previously typed as Rh-negative) and placental blood tested over a period of three months was undertaken. The number totalled 204, and of these it was found that only 46 (22·5%) were the same blood group and Rh type, whilst 83 (40·7%) were the same group and different Rh type, 46 (22·5%) of different group and different Rh type, and 29 (14·2%) of different group and the same Rh type.

From this analysis it does not appear that the numbers of maternal and placental specimens of blood of the same group and Rh type are disproportionately great. To clarify this point further, blood was taken from 20 babies whose placental blood group and Rh type were identical with those of their mothers, and their blood was checked. In each case there was no change of result.

Thus it appears that there is little or no mixing of maternal blood with placental blood in the cord, and the testing of placental blood for the baby's Rh type and blood grouping is quite justified.

Reports of Cases.

ANEURYSM OF THE RIGHT INTERNAL ILIAC ARTERY: FIVE YEARS' CURE.

By KEITH KIRKLAND and KENNETH W. STARR,
Sydney.

ON September 16, 1947, H.R.J., a male patient, aged fifty years, was referred to one of us (K.K.) by Dr. E. Huth and stated to have an enlarged prostate with pulsation. The patient complained of increasing difficulty of micturition for the preceding six months, and said that his urinary stream was then very poor. He also complained of dysuria, frequency of micturition and the necessity to pass urine two or three times during the night. He had suffered intense pain in the right groin, and this appeared to radiate to the right sacro-iliac joint. The pain was made worse by sitting down and relieved by bowel action. There was no history of diarrhœa. Right epididymitis had occurred eighteen months previously without urethritis and had settled spontaneously. The administration of an œstrogen for one month had not in any way relieved the patient.

The patient was a fat, hypertensive male; his weight was 13 stone seven pounds, and his blood pressure was 190 millimetres of mercury, systolic, and 130 millimetres, diastolic. Rectal examination suggested an enlargement of the right lobe of the prostate, which was of firm consistency but pulsating. This pulsation appeared to be transmitted from a large right pelvic mass. When the patient passed urine, the pulsation was noticeable in the stream. A pistol-shot sound was audible in the groin and below and to the right of the navel. The femoral pulse was normal, and a provisional diagnosis of aneurysm of the right internal iliac artery was made. Both the Wassermann test and the Kahn test produced negative results. A radiographic examination of the lumbo-sacral part of the spine and of the pelvis and femora on September 24 revealed the following abnormalities. Congenital spondylolisthesis was present, with defect of the lamina of the fifth lumbar vertebra; the latter was displaced forward in relation to the sacrum. The part of the lumbar section of the spine composed of the third, fourth and fifth vertebræ was displaced forward as a whole, and this displacement was in relation to the second lumbar vertebra above, with considerable widening of the apophyseal joint between the third and fourth lumbar vertebræ. The joint above was similarly widened. There was a thinning of the disk space between the second and third lumbar vertebræ, and between the fifth lumbar and first sacral vertebræ, due to degenerative changes. No abnormality was detected in the pelvis or in the upper portions of the femora.

A cystoscopic examination was attempted on October 2, but it was found impossible to introduce the instrument. The urethra was injected with radio-opaque solution and an informative cysto-urethrogram was obtained. The bladder and the proximal part of the urethra were shown to be displaced to the left and apparently anteriorly. The bladder was reduced to a crescentic shape by a large filling defect on the right side.

On November 4, with the patient under "Pentothal"-tubarine-ether anæsthesia, a right extraperitoneal exposure of the aneurysm was effected through a six-inch incision. The sac was about the size of a small football and filled the pelvis. About one inch of normal internal iliac artery preceded the aneurysmal sac and intervened between the saccular aneurysm and the common and external iliac arteries. Some difficulty was encountered in passing an aneurysm needle above this segment of the vessel, and the sac wall was torn, with rather alarming hæmorrhage. However, this was readily controlled by finger pressure until the double number 16 silk ligature was tied. Closure and convalescence were uneventful. Relief of urinary symptoms after operation was dramatic and immediate.

In May, 1949, when reexamined, the patient had a free urinary stream which was without pulsation, and nocturia had disappeared. The prostate felt normal on rectal examination and no pulsation could be detected. He had no urinary symptoms when examined in December, 1950, for an injury to the medial meniscus of his right knee.

In March, 1952, dysuria was present with occasional hæmaturia, and he was further investigated. The hæmaturia was painless and the blood appeared to be intimately mixed with the urine; the attack had occurred three weeks before, and been followed by severe pain in the right groin. Excretion urography revealed no calculi in the urinary tract; there was some calcification of the pelvic vessels. Both kidneys secreted the dye satisfactorily. The bladder appeared to be rather small and spastic.

In April, 1952, a great deal of pus was present in the prostatic bead. Five prostatic massages were carried out at weekly intervals, with considerable benefit. A cystoscopic examination was advised, but was deferred by the patient.

In March, 1953, the patient reported that he was feeling well and had had no further bleeding or symptoms.

Comment.

This case illustrates an unusual cause of prostatic obstruction, and the long-term relief derived from proximal ligation of the right iliac artery.

SUDDEN COLLAPSE AND DEATH IN INFANTILE ECZEMA: REPORT OF A CASE.

By C. R. BOUGHTON, M.B., B.S.,
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IT is perhaps not sufficiently realized that admission to hospital of babies suffering from infantile eczema may seriously jeopardize their chances of survival (Twiston Davies, 1940). During the first two weeks in hospital the child with infantile eczema runs considerable risk of sudden collapse, which may terminate fatally for no apparent reason and even at a time when the skin condition appears to be improving. The case reported below illustrates several of the recognized factors involved in these catastrophes and emphasizes the care with which these patients should be treated.

Clinical Record.

The patient was a male baby, aged eight weeks, the first born of an Rh-negative woman. Inquiry into the family history showed that although neither the child's mother nor his father suffered from apparent allergic disorders, a nephew of the child's mother suffered from severe asthma and also an aunt on the mother's paternal side. Labour had been normal, and the child's birth weight was nine pounds one ounce. The child was breast fed from birth and took his feeds well. Regurgitation of some of each feed occurred regularly within fifteen minutes of ingestion. He regained birth weight by the age of three weeks, and continued to gain until the sixth week, when he weighed nine pounds 12 ounces. At this time vomiting became more severe and the weight fell eight ounces during the period from the sixth to eighth week of life. The vomiting was never projectile. The bowels moved every three days and the stools were apparently normal. At the age of two weeks he developed a rash on the face and head which spread to the trunk and thighs. He was admitted to hospital at the age of eight weeks with a provisional diagnosis of possible diaphragmatic hernia or possible pyloric stenosis.

Physical examination revealed the baby to be fretful and underweight; he had an extensive weeping eczematous rash on the head and cheeks, in the flexures of the arms, in the groins and behind the knees (Hetreed, 1949; Glaser, 1948). The pulse rate was 140 beats per minute; the pulse was regular in rhythm and constant in amplitude. No intraabdominal swellings were felt. The temperature was 100° F. Other findings were essentially normal.

A blood count on the day of his admission to hospital gave the following information: the hæmoglobin value was 12·0 grammes *per centum*, the white cells numbered 11,000 per cubic millimetre, and the differential leucocyte count was normal. Examination by means of a barium meal revealed no abnormality in the œsophagus, stomach or duodenum, and the meal reached the ileum in two hours. No evidence of diaphragmatic hernia was found. An X-ray examination of the chest revealed no abnormality.

The baby was fed at first on expressed breast milk (Grulee, 1936; Hill, 1947; Brain, 1952) with "Glaxo Number 1" as complement. Zinc cream and castor oil were applied to the flexures and head. Boric lotion was not used because of the extent of the weeping surfaces and the probability of increased absorption of the compound (Watson, 1945). Four days later feeding was changed to breast milk together with half-strength "Sunshine" dried milk, with the addition of 12 teaspoonfuls of sugar to the pint. The skin condition improved, but two days later the vomiting after meals increased in severity and increasingly rapid loss of weight became apparent. The feeding was suspended and 5% glucose and half normal saline was given by mouth every two hours, preparatory to the child's being weaned on to soya bean milk or goat's milk in the event of the vomiting ceasing. This feeding had been instituted only some hours when the child suffered a sudden attack of collapse in which the extremities became cold and pale and respiration was gasping. The pulse was

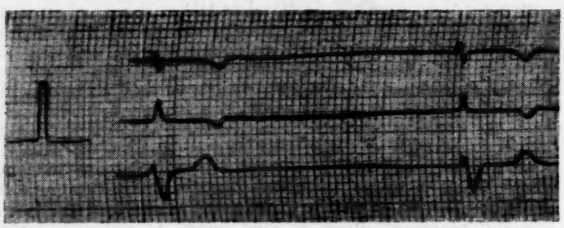

FIGURE I.

Electrocardiograph (portable machine) showing standardization and leads I, II and III. The leads are not recorded synchronously. The tracing shows complete heart block, widening of the *QRS* complex, prolonged *Q-T* interval, inverted *T* waves in leads I and II with idioventricular rhythm. The voltages generally are low.

imperceptible and the heart rate was found to be 30 per minute. The rhythm was regular. A systolic murmur was reported to the left of the sternum, and the second heart sound was split. Respiration became periodic, and terminally rapid (50 per minute) and shallow. The temperature was 97° F.

Ephedrine sulphate and *Liquor Adrenalinæ* (one in 1000) were administered hypodermically at intervals of a quarter to half an hour, with temporary improvement. Oxygen was given by nasal catheter and a glucose-saline infusion was commenced.

An electrocardiogram was taken at this time. No *P* waves were seen, the duration of the *QRS* complex in lead III was 0·16 second, the *Q–T* interval was 0·55 second, and T1, T2, TAVL, TAVF were inverted and 0·12 second in duration. The *R* wave in lead I was 0·15 millivolt and the *S* wave in lead III was 0·46 millivolt. The appearances were those of complete heart block with idioventricular rhythm, intraventricular block and prolonged *QT*; the findings were consistent with gross electrolyte imbalance including hypokalæmia (Lipman, 1952; Grabiel and White, 1947; McAllen, 1951; Katz, 1947; Sollman, 1947; Thomson, 1939; Stewart *et alii*, 1940; Stoll and Nisnewitz, 1941), or anoxia.

Accordingly a stomach tube was passed and potassium citrate (10 grains) was given in solution. This dose was

repeated in two hours' time, but apparently had no effect on the cardiac dysfunction. The child died twelve hours after the onset of the attack.

At autopsy no gross abnormality was found. No atrial or ventricular septal defects were present; the gastro-intestinal tract, including the pylorus, was normal. General and pronounced œdema of the cerebral tissue was found on microscopic examination.

The fact that sudden death may take place in the course of infantile eczema has been recognized for many years (Twiston Davies, 1940; Paterson and Moncrieff, 1949; Schwartz, 1950; Sheldon, 1951). The sudden collapse has been variously ascribed to *status thymolymphaticus*, to overwhelming infection, to acute anaphylactic shock, or even to an acute psychological reaction to separation from the mother. It has been noted that the most dangerous time is the first seven to fourteen days in hospital (Twiston Davies, 1940). It is stated that very rarely, if ever, does a child with infantile eczema die in its mother's arms, and for this reason many pædiatricians refuse to accept such patients for admission to hospital.

The catastrophe often occurs when the skin condition is apparently improving; it occurs most frequently in the first year of life, and often in fat infants with or without a tendency to rickets. Seldom does autopsy reveal a satisfactory cause for the collapse. In this particular case the child was aged eight weeks; considerable vomiting had occurred with loss of weight, producing some degree of dehydration and certainly a pronounced electrolyte disturbance (Wright, 1952). It has been stated that transitory pylorospasm in the newborn may be allergic in origin, and that the same child will often show other allergic responses subsequently (Santillana, 1950). Furthermore, severe sodium chloride deficiency may give rise to vomiting probably also due to pylorospasm (Wright, 1952).

The collapse, as is usual in such cases, was quite sudden, on the eighth day in hospital, at a time when considerable improvement in the skin condition had occurred. The baby was pale and looked shocked, with slow gasping respiration, leading later to Cheyne-Stokes respiration, and finally rapid shallow respiration. In some cases the cause of the sudden deterioration is probably acute anaphylactic shock, a common precipitant being egg white (Hill, 1947). Any severe infection such as pneumonia or gastro-enteritis (Paterson and Moncrieff, 1949) can be rapidly fatal in such a child; however, the occurrence of complete heart block with the interesting electrocardiographic findings, similar reports of which could not be found in the literature, has prompted the thought that perhaps in some cases of infantile eczema sudden death may be due to severe electrolyte disturbance, probably with potassium and calcium ion deficiency, terminating in acute circulatory collapse with anoxia and attendant damage to the sensitive conducting tissues of the heart. The baby with infantile eczema can lose circulating fluid very rapidly in vomiting or diarrhœa (Darrow, 1946), from the extensive weeping areas which cover a considerable fraction of the total body surface, and also into the tissues in response to scratching or on contact with any substance to which it is allergic, and which may be absorbed through the broken or intact skin (Hill, 1947). These various factors may profoundly disturb the already delicate fluid balance of a child in the first year of life. Furthermore, administration of a glucose-saline solution will tend to depress further an already low serum potassium level (Hawkins *et alii*, 1951).

The administration of Ringer's solution or serum by intravenous infusion would seem to be the most logical form of replacement therapy when vomiting is occurring, supplemented if necessary by the administration of additional potassium and calcium salts. Butler's solution (Wright, 1952) and Elkinton's solution (Wright, 1952) are preparations containing potassium, but great care is needed in administering potassium salts parenterally because of the risk of sudden cardiac arrest (Howard and Carey, 1949). Adrenaline, ephedrine and oxygen may support the circulation and vital functions and combat the tendency to heart block until effective replacement treatment has been instituted. Suprarenal cortical extract has been used with some success (Marsden, 1947).

Summary.

1. A case of sudden collapse and death in infantile eczema is reported.

2. The suggestion is made that death was due to acute electrolyte imbalance.

3. It is stressed that these babies should always be watched carefully and not admitted to hospital unless absolutely necessary.

Acknowledgements.

I am grateful to Dr. Wallace Freeborn, General Medical Superintendent of the Royal North Shore Hospital, for permission to publish this case report. I wish to express my sincere thanks to Dr. Frank Lawes, Honorary Physician, under whose care the patient was admitted to hospital, and to Dr. Clair Isbister, Honorary Assistant Pædiatrician, who very kindly saw the patient in consultation, for her invaluable assistance and criticism.

References.

BRAIN, R. T. (1952), "The Problem of Eczema in Infancy and Childhood", *Postgrad M. J.*, 28 : 165.
CRESPO-SANTILLANA, A., and LLOPIS REY, J. J. (1950), "Allergic Manifestations Due to Feeding Encountered During Infancy", *Acta pediat. espan.*, 87 : 259; quoted in *Am. J. Dis. Child.* (1951), 82 : 649.
DARROW, D. C. (1946), "Retention of Electrolyte During Recovery from Severe Dehydration Due to Diarrhœa", *J. Pediat.*, 28 : 515.
GLASER, J. (1948), "Treatment of Eczema (Atopic Dermatitis) in Infancy", *J.A.M.A.*, 137 : 527.
GRAYBIEL, A., and WHITE, P. D. (1947), "Electrocardiography in Practice", Second Edition, Saunders, Philadelphia.
GRULEE, C. G., and SANFORD, H. N. (1936), "The Influence of Breast and Artificial Feeding on Infantile Eczema", *J. Pediat.*, 9 : 223.
HAWKINS, C. F., HARDY, T. L., and SAMPSON, H. H. (1951), "Potassium Deficiency in Pyloric Stenosis", *Lancet*, 1 : 318.
HETREED, F. W. (1949), "Management of Atopic Dermatitis (Eczema)", *Med. Clin. North America*, 1 : 205.
HILL, L. W. (1947), "The Pathogenesis of Infantile Eczema", *J. Allergy*, 18 : 60.
HOWARD, J. E., and CAREY, R. A. (1949), "Use of Potassium in Therapy", *J. Clin. Endocrinol.*, 9 : 691.
JOLLMAN, T. (1948), "A Manual of Pharmacology", Seventh Edition, Saunders, Philadelphia.
KATZ, L. N. (1947), "Electrocardiography", Second Edition, Lea and Febiger, Philadelphia.
LIPMAN, B. S., and MASSIE, E. (1951), "Clinical Unipolar Electrocardiography", Year Book Publishers Inc., Chicago; 138.
MCALLEN, P. M. (1951), "The Electrocardiogram Associated with Low Levels of Serum Potassium", *Brit. Heart J.*, 13 : 159.
MARSDEN, C. W. (1947), "Suprarenal Cortical Extract for Collapse in Infantile Eczema", *Brit. M. J.*, 1 : 808.
PATERSON, D., and MONCRIEFF, A. (1949), "Diseases of Children", 2 : 951.
SCHWARTZ, A. B. (1950), "Deaths in Hospitalized Eczema in Infants", *Arch. Pediat.*, 67 : 295.
SHELDON, W. (1951), "Diseases of Infancy and Childhood", Sixth Edition, Churchill, London; 679.
STEWART, H. J., SMITH, J. J., and MILHORAT, A. T. (1940), "Electrocardiographic and Serum Potassium Changes in Familial Periodic Paralysis", *Am. J. M. Sci.*, 199 : 789.
STOLL, B., and NISNEWITZ, S. (1941), "Electrocardiographic Studies in a Case of Periodic Paralysis", *Arch. Int. Med.*, 67 : 755.
THOMSON, W. A. (1939), "Potassium and the T Wave of the Electrocardiogram", *Lancet*, 1 : 808.
TWISTON DAVIES, J. H. (1940), "Sudden Death in Infantile Eczema", *Brit. J. Dermat.*, 52 : 182.
WATSON, E. H. (1945), "Boric Acid. A Dangerous Drug of Little Value", *J.A.M.A.*, 129 : 332.
WRIGHT, S. (1952), "Applied Physiology", Ninth Edition, Oxford, London; 65, 109, 971.

A CASE OF SPONTANEOUS RUPTURE OF THE SPLEEN.

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Clinical Record.

A.G., A LUMPER, aged forty-six years, was admitted to the Royal Adelaide Hospital at 3 a.m. on July 29, 1945, complaining of severe pain extending from his left shoulder

down the left side of the trunk. The history obtained from the patient was that he came home from work on the night before, played cards early in the evening, and was suddenly seized with severe pain in the left side when he went to bed at 9 o'clock. The pain was continuous until his admission to the hospital. On examination, he was found to be in a state of partial collapse; his temperature was 96° F., his pulse rate was 72 per minute, and his respirations numbered 18 per minute. He had board-like rigidity in the region of the left hypochondrium, Kehr's sign was present and Ballance's sign was absent (the reason will be obvious later). His blood pressure was 105 millimetres of mercury, systolic, and 80 millimetres, diastolic. The only relevant detail in his past history was that he had had malaria at about the time of the last war. A provisional diagnosis of ruptured peptic ulcer was made, and arrangements were made for operation. His condition remained fair during the rest of the night; his temperature was 98·4° F., his pulse rate was 120 per minute, and his respirations numbered 22 per minute at 8 a.m.

At 8.30 a.m. on the same day, laparotomy was performed under a general anæsthetic; a right paramedian incision was made, extending from the level of the umbilicus five inches upwards. The stomach and duodenum were found to be normal, but when the stomach was moved to the right large quantities of blood clot were found in front of the spleen and over the upper pole of the right kidney and extending upwards under the left cupola of the diaphragm. When approximately one pint of this had been removed, the spleen was found to have a circular rent around its upper pole, the bare area being covered by a layer of blood clot. The spleen was then stripped of its ligamentous attachments and the pedicle tied in three stages. The tail of the pancreas being unimpaired, hæmostasis was secured, the abdomen was closed at 9.10 a.m. and the transfusion of a pint of blood instituted before the patient left the operating theatre. Later a further pint of serum followed by saline and glucose was given through the drip apparatus. The condition of the patient throughout and after the operation was reported by the anæsthetist as fair; his temperature was 97° F., his pulse rate was 120 per minute, and his respirations numbered 14 per minute.

On macroscopic examination, the capsule of the spleen was found to be wrinkled and the organ of apparently normal size. Microscopic examination revealed a normal structure with fresh hæmorrhages.

On the second day the patient developed left lower lobar pneumonia, which responded well to sulphadiazine, his chest being clinically clear in six days. Radiological examination on the eighth day revealed no abnormality. Apart from the pneumonia, his recovery was uneventful.

Comment.

As only about 35 cases of this condition are recorded in the literature, it seemed worthy of description. When the literature on the subject is reviewed, there seems to be much justifiable doubt on the possibility of its being an absolutely unprecedented mishap, and some of the cases are not without history of what might have been relevant factors in the causation of the condition. Grossmann (1942), writing of the same condition, makes reference to the fact that "the occurrence of a non-traumatic rupture of a previously normal spleen has been known for centuries", while at the same time he states that "perhaps a total of two or three dozen cases have been cited in the literature of the present time". In proffering a classification of non-traumatic rupture, he makes subdivision into two categories: (i) chronically diseased spleens, (ii) acutely diseased spleens. The former group are those likely to be classified as primary non-traumatic rupture, and Brines (1943) points out that it is quite probable that the site of rupture may be one abnormal portion of the organ while in the remainder the tissue is normal. The same writer goes on to point out that such conditions as coughing, sneezing, twisting and lifting may so increase the intraabdominal tension as to be potent causes in bringing about rupture of the spleen. It may therefore be significant to point to the occupation of the patient whose case is quoted above. Watson and Frederer (1942) quote the case of a patient with the same condition who was admitted to hospital with slight bruising of the dorsum of one hand and elbow and about the left eye. Here is an instance of what appears superficially to have been a mild trauma. Susman (1927) believed four conditions of the spleen to be likely causative factors: (*a*) softening of the spleen and capsule; (*b*) congestion of the portal vein; (*c*) formation of a subcapsular hæmatoma; (*d*) perisplenic adhesions.

It is perhaps worthy of mention that Lundell (1934), who studied reports of 20 of the cases, is not satisfied of their absolutely non-traumatic nature. Be this criticism as it may, the above case is presented in the belief that no previous injury of a day or some weeks' duration could be invoked as a causative factor.

Acknowledgement.

My thanks are due to Dr. R. A. Barter, who wrote the above commentary for me when he was a fifth year medical student.

References.

BRINES, O. A. (1943), "Spontaneous Rupture of Normal Spleen", *Arch Path.*, 36 : 163.

GROSSMANN, L. L. (1942), "Nontraumatic Rupture of Previously Normal Spleen and its Medicolegal Aspect; Clinical and Pathological Report Revealing Embolic Manifestations of an Apparently Insignificant Traumatic Injury", *Wisconsin M. J.*, 41 : 477.

LUNDELL, G. (1943), *Acta chir. scandinav.*, 75 : 59.

SUSMAN, M. P. (1927), "Spontaneous Rupture of Spleen", *Brit. J. Surg.*, 15 : 47.

WATSON, J. R., and FREDERER, M. (1942), "Spontaneous Rupture of the Normal Spleen", *J.A.M.A.*, 120 : 690.

Reviews.

A Handbook of Radiotherapy for Senior and Post-Graduate Students. By Walter M. Levitt, M.D., F.R.C.P. (London), F.F.R., D.M.R.E. (Cambridge), of Lincoln's Inn, Barrister-at-Law; 1952. London: Harvey and Blythe, Limited. 9" × 5½", pp. 224, with 53 illustrations. Price: 30s.

Physical Foundations of Radiology. By Otto Glasser, Edith H. Quimby, Lauriston S. Taylor and J. L. Weatherwax; Second Edition; 1952. New York: Paul B. Hoeber, Incorporated. 7½" × 5½", pp. 594, with 112 text figures. Price: $6.50.

Two books come up for review and they may well be considered together, for although they differ greatly in subject matter, the fundamental purpose behind each of them is the same.

"A Handbook of Radiotherapy for Senior and Post-Graduate Students" by Walter M. Levitt deals with radiotherapy from the clinicians' viewpoint only, avoiding all technical discussion and detail of interest only to the professed radiotherapist, whilst "The Physical Foundations of Radiology" is an exposition of radiological physics on a descriptive plane, suited to the limited mathematical equipment of the radiologist. This book completely avoids the thin air of higher mathematics so beloved of the physicist.

Both books, therefore, may be described aptly as liaison books, interpretative in character for the use of lower, or perhaps it is better to say less specialized, levels of scientific intelligence. They present the essential principles of applied radiotherapy for the clinician and of applied physics for the radiologist respectively. They are therefore both welcome gifts to the library of practical, as opposed to purely scientific, knowledge.

Such books are immensely valuable in a world where the centrifugal forces of intensive specialism are bursting the wheel of knowledge into thousands of separated fragments. Their worth, of course, depends upon the detailed scientific knowledge of the authors, and in both the instances under discussion such sources are beyond reproach. The selection and integration of the materials considered essential have been made with nice judgement and evident appreciation of the needs of the practical practitioner in either branch. Both communications are a realization of the principle that one of the first duties of the specialist is to interpret the results of his specialism in terms of plain English devoid of

technical jargon and mathematical farrago. The faint-hearted can thus pursue their studies into foreign fields without learning a new language and the initiated can read for rapid revision without confusion or boredom. The super specialist thus fulfils what is probably his most important function and disseminates the concrete achievements of his researches for practical application. This is scientific cooperation in its best form.

In "A Handbook of Radiotherapy" by Walter Levitt, the first half of the book is devoted to general principles. There is a succinct statement of the physics of radiation and its application to the patient and of the fundamentals upon which treatment is based. One particularly useful chapter for the referring practitioner is that on the management of the patient during and after irradiation. The second half of the book deals with the treatment of disease system by system and disease by disease. This is a fair and useful statement of the present-day indications for radiotherapy and of the results which may be reasonably expected. Malignant conditions, of course, bulk largely in this section and carcinoma of the breast is fairly and fully treated. Other non-malignant conditions, however, such as spondylitis and affections of the eye, also are included. This book should be read and digested by every general physician and will give an intelligent understanding of the uses of irradiation in medicine with interest and without undue effort. It is eminently sane, practical and devoid of specialist bias.

"The Physical Foundations of Radiology" by Glasser, Quimby, Taylor and Weatherwax needs little introduction. It is a second edition of a well-known and well-appreciated work of many years' standing. The subject of radiation physics is constantly expanding and therefore it has been necessary to rewrite many of the chapters. Radioisotopes are adequately covered in two additional chapters and the information on protection from irradiation has been brought up to date in the light of recent researches. This is an excellent text-book, expanded to include recent activities and maintaining all its previous superlative qualities.

The production of both editions is beyond reproach, with clear-cut legible type, good paper and tasteful bindings—small factors which, strange to say, make a book more interesting irrespective of the subject matter.

Posture and Pain. By Henry O. Kendall, Florence P. Kendall and Dorothy A. Boynton; 1952. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 11½" × 9", pp. 212, with 159 illustrations. Price: 75s. 3d.

THIS book merits a place in the library and physiotherapy department of every hospital. The work of the Kendalls, of Baltimore, has already earned the gratitude of doctors and physiotherapists interested in muscle function, especially those responsible for the after-care of poliomyelitis patients. For twenty-eight years Henry O. Kendall has been in charge of the Physiotherapy Department of the Children's Hospital School, Baltimore, and, with his wife, Florence Kendall, has been associated with the work of the orthopædic division of Johns Hopkins Medical School. In 1938 a small book published as United States Public Health Bulletin No. 242 summarized the experience of the Kendalls in testing for muscle function. This bulletin has been an accurate guide and text-book for physiotherapy students in many centres. In 1948 the Kendalls completed a large book, "Muscle Testing and Function", in which 162 large and clear illustrations have made easy the task of the student seeking knowledge of muscle action. "Look it up in Kendall's book" has become the answer to any argument concerning paresis of muscles. Last year, in a companion volume, "Posture and Pain", Mr. and Mrs. Kendall, with the help of Dorothy Boynton, set forth the fruit of two lifetimes of experience. They have brought to the study of body mechanics the careful analysis of muscle action which has characterized their years of work to help the patients whom they handled. The motive for the book springs from a recognition of the prevalence of postural faults, the associated pain and the subsequent waste of human resources. If this excellent book merits any criticism, one would suggest that the section "Prevention of Postural Faults" could be expanded to include more details concerning posture in infancy and during the pre-school years. Readers who own the earlier book, "Muscle Testing and Function", would have welcomed replacement of illustrations produced therefrom by more illustrations in the section on prevention—illustrations which would guide parents on the hazards, for example, of the child writing with the left hand, or spending hours practising the piano-accordion; or which would influence the adult patient in choosing desirable postures in lifting or pushing heavy objects. However, other writers can produce this type of public education, but only workers who have studied one field of medicine with tireless energy and persistence could have produced the text and arranged the excellent line drawings and photographs of this book.

An Atlas of the Commoner Skin Diseases: With 147 Plates Reproduced by Direct Colour Photography from the Living Subject. By Henry C. G. Semon, M.A., D.M. (Oxon.), F.R.C.P. (London), colour photography originally directed by the late Arnold Moritz, B.A., M.B., B.C. (Cantab.); Fourth Edition; 1953. Bristol: John Wright and Sons, Limited. 10" × 7½", pp. 380, with 147 plates in colour. Price: 75s.

THIS excellent work has been enlarged by the inclusion of eight new plates. These are equal in quality to the older reproductions and all reflect credit on the art of clinical photography as a medium of instruction and reference. The atlas will hold its place as a first class basis for the teaching of dermatology. It is of great use also to the specialist dermatologist and to the general practitioner. The text is admirably condensed and very readable.

Treatment covers briefly all essentials and is sound in principle. However, it is doubtful if many Australian dermatologists have had as much success in the treatment of tinea with undecylenic acid as Dr. Semon appears to have had in England. The author deserves great praise for his repeated references to the risk of epidermal sensitization and its sequelæ likely to follow unrestricted local use of sulphonamides and antihistamines. This matter was the subject of a recent letter in this journal by the Dermatological Association of Australia. These agents are used infrequently and with great caution by informed Australian practitioners.

The advice given to medical witnesses at workers' compensation courts is worthy of note. It is pointed out that the practitioner's presence is merely to help the court. Sides should not be taken. Primary mycotic dermatitis affecting the fingers and hands is not regarded as very common in Australia. It appears to be commoner in England. The diagnosis requires cultural confirmation. Timely reference is made to the occurrence of perianal dermatitis and stomatitis due to aureomycin therapy. The use of the vitamin *B* group with this antibiotic is strongly advised.

The atlas is well arranged and well produced and can be strongly recommended to the dermatologist, general practitioner and student. We deeply regret the death of Dr. Semon's friend and collaborator. Dr. Arnold Moritz.

Surgery for Students of Nursing. By John Cairney, D.Sc., M.D., F.R.A.C.S., with a foreword by Miss M. I. Lambie, C.B.E.; 1952. Christchurch: N. M. Peryer, Limited. 9" × 6", pp. 334, with 120 text figures. Price: 40s.

UNLIKE most books on surgery for nurses, this book is not intended to cover surgical nursing; rather it would form a basis for it. While it has been primarily written for nurses in New Zealand, it will be found to be of considerable use to nurses in other countries. The text is well set out, and the drawings (by the author himself) are clear. Nurses who have difficulty in obtaining access to catalogues of surgical instruments would not agree that illustrations of instruments and appliances are better omitted. Also, some apparatus, such as that for transfusions, gastric aspiration, or tidal drainage, is rarely included in instrument catalogues. Another minor criticism that may be made of what is otherwise an excellent book for its purpose is the inclusion of the nearly obsolete Pilcher's bag, the Stamm type of gastrostomy, and the rubber dam and gauze packing for the perineal wound after excision of the rectum. There is no mention of the retropubic prostatectomy. However, the book more than covers the field of surgery for nurses, there are very few typographical errors (an exception being "obstructuve" on page 152), and it has been kept a reasonable size. It may therefore be thoroughly recommended as a book of study and reference for nurses.

Fundamentals of Clinical Orthopedics. By Peter A. Casagrande, M.D., and Harold M. Frost, junior, M.D., with forewords by Joseph S. Barr, M.D., and Frank N. Potts, M.D.; 1953. New York: Grune and Stratton, Incorporated. 11" × 8½", pp. 592, with 390 illustrations. Price: $18.50.

THIS book has a very wide range, a fact acknowledged by the authors. By the enlisting of able aid from many contributors in special subjects, the whole field of orthopædics has been covered. It is considered that this aim has been too ambitious and the authors would have been well advised to omit some subjects—notably fractures. In this way other aspects could have been enlarged upon.

The presentation of the subject matter is not so detailed as is required in a text-book, nor yet is it sufficiently condensed to be termed a synopsis. The omission of any bibliography is a major fault, as further study is essential to full mastery of many of the conditions dealt with.

Apart from this general criticism, there are many features about this book which are excellent. The presentation and grouping of the subject are unusual, but are especially good. The first section is termed "Basic Science" and sets out clearly and concisely the modern physiological concepts of tissue and bone metabolism. To those who graduated some time ago this will undoubtedly appeal.

The section on "Affections of Bone" repays study. Here the various bone disorders are divided, according to ætiology, into four groups, namely, the deficiency diseases, the vascular disorders, the infective conditions, and miscellaneous varieties.

Another excellent section is that on "Amputations, Prosthetics, Braces, Shoes, and Shoe Corrections". The actual fitting of prostheses and the actual design of a shoe with its alteration for orthopædic conditions are too often neglected in many standard text-books.

The book itself is well produced and set in good clear type with sufficient well-chosen illustrations and X-ray reproductions. It is a praiseworthy attempt to supply a ready reference book on orthopædic conditions. As such it should appeal to resident medical officers and post-graduate students. For the latter, however, it should serve as a preliminary to wider study and reading.

Reason and Unreason in Psychological Medicine. By E. B. Strauss, M.A., D.M. (Oxon.), F.R.C.P., with a foreword by Russell Brain, M.A., D.M. (Oxon.), P.R.C.P.; 1953. London: H. K. Lewis and Company, Limited. 8¼" × 5", pp. 68. Price: 8s. 6d.

THIS small book of three chapters is written in lecture form. Attention is directed towards the "middle way" in psychiatry. The nature of the mind in relation to the brain is considered afresh in the light of the new knowledge of psychophysiology and of the new approach to logic. There are closely argued points which represent the author's impressions of progress and development in the psychiatric movement. The relationship of the body, the psyche, and the soul is discussed. The author has no quarrel with psychoanalytical methods, but only with the claims that they penetrate religion, philosophy, education and art. He criticizes the sweeping and all-embracing but immutable doctrine of Freudian philosophy. Under the best conditions for treatment the 25% of cures leaves much to be desired and much to be explained.

The second and third chapters are the Croonian Lectures of 1952 which were delivered before the Royal College of Physicians. The first of these is a challenge and critical assessment of our easy acceptance of causality in reference to medical reasoning. The author points out that much of what we are prepared to accept as causation is no more than an evasion of the question. A contrast is developed between this and the classical forms of reasoning.

This book is an interesting and thoughtful work with a critical approach. It will be very helpful to many who, like the author, do not feel satisfied with the current approach and reasoning in psychological medicine.

The Singer's and Actor's Throat: The Vocal Mechanism of the Professional Voice User and its Care in Health and Disease. By Norman A. Punt; 1952. London: William Heinemann (Medical Books), Limited. 9" × 6", pp. 100, with four text figures. Price: 10s.

TO explain in simple form the vocal mechanism of the professional voice user, and its care in health and disease, is the purpose of Mr. Norman A. Punt's little book "The Singer's and Actor's Throat". The book is intended mainly for singers, lecturers, public speakers and actors, and for their teachers. The aim has been to describe the mechanism of speech and of singing and then to show how incorrect methods, over-straining, forcing beyond reasonable natural capacity, singing or speaking in the presence of inflammatory conditions *et cetera* may cause permanent damage. A brief summary is given of the features which are essential for the production of a pleasing singing or speaking voice. There is a brief outline of the most desirable anatomical qualities. The psychological background which may cause fear and apprehension in a singer anxious about his or her voice is sympathetically described so that the great value of reassurance by the laryngologist is well able to be appreciated. In a discussion on care and use of the voice in health, "glottic shock" or forcing to reach a pitch beyond the natural capacity is severely condemned. Excessive muscular tension must be avoided and singing should be confined to roles within the natural range of the individual. Singing should never be too loud, nor too long or often. Excessive and fatiguing practice is both unnecessary and harmful. In all cases of voice weakness and in inflammatory conditions the need for voice rest for adequate periods and for very gradual resumption thereafter is stressed. Mild sprays and gummy saliva-stimulating lozenges may help to tide the patient over an emergency. The author reveals no secret remedies. The actor, singer or public speaker will at least understand how his voice is produced after reading this little book and he will know a little of how to determine his range and limitations. He should finish it knowing how to avoid self-inflicted damage to his own singing or speaking capacity. The complexity of the problem, however, is suggested by an imposing bibliography of 88 publications and references.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Surgical Clinics of North America"; 1953. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. New York Number. 9" × 6", pp. 316, with 102 illustrations. Price: Paper bound, £6 *per annum*; cloth bound, £7 5s. *per annum*.

The number consists of twenty contributions to a symposium on the surgery of cancer. There are two clinics on other subjects. This is a New York Number.

"Some Problems in Neurophysiology", by F. Bremer; 1953. London: University of London, The Athlone Press. Sydney: Walter Standish and Sons. 9" × 5½", pp. 88, with 32 illustrations. Price: 21s.

The book had its origin in three lectures delivered before the University of London.

"Modern Medical Monographs: Chronic Pulmonary Emphysema: Physiopathology and Treatment", by Maurice S. Segal, M.D., and M. J. Dulfano, M.D.; 1953. New York: Grune and Stratton. 9" × 6", pp. 190, with 31 illustrations. Price: $5.50.

Based on a series of lectures on the treatment of chronic pulmonary emphysema in which laboratory research and the results of experience were combined.

"Hernia: A Manual for Truss Fitters", by Francis Mitchell-Heggs, T.D., M.B., B.S. (London), F.R.C.S. (England), F.R.C.S. (Edinburgh), with forewords by Cecil Wakeley, K.B.E., C.B., F.R.C.S. (England), F.A.C.S., F.R.A.C.S., and Claude Frankau, C.B.E., D.S.O., M.S., F.R.C.S. (England); 1953. London: J. and A. Churchill, Limited. 10" × 7½", pp. 144, with 97 illustrations. Price: 25s.

The syllabus from which the book was written was compiled by a committee of the Royal College of Surgeons of England.

"An Approach to Clinical Surgery", by Gerald H. C. Ovens, O.B.E., M.B., B.S. (London), F.R.C.S. (England); 1953. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 310, with 118 illustrations. Price: 22s. 6d.

The title describes the object of the book; it is divided into two parts—"General Principles" and "History-Taking and Examination"—and there are several chapters of "special schemes".

"The Physician in Atomic Defense: Atomic Principles, Biologic Reaction and Organization for Medical Defense", by Thad P. Sears, M.D., F.A.C.P., with a foreword by James J. Waring, M.D., M.A.C.P.; 1953. Chicago: The Year Book Publishers, Incorporated. 8" × 6", pp. 308, with 53 illustrations. Price: $6.00.

This book aims at a more rational understanding of the problems and applications of atomic energy.

"Social Service and Mental Health: An Essay on Psychiatric Social Workers", by Margaret Ashdown and S. Clement Brown; 1953. London: Routledge and Kegan Paul, Limited. Sydney: Walter Standish and Sons. 9" × 5½", pp. 268. Price: 16s.

This book is about those "who choose and are chosen for psychiatric social work, the use they make of training and the shaping of their careers".

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

HENRY WELLCOME: A CENTENARY.

On August 21, 1853, in a log-cabin in Wisconsin, United States of America, where his father was a missionary among the Dakota Indian tribes, Henry Wellcome was born. This child was destined to take a prominent place in the world of science and to leave a name which will be honoured wherever medicine is studied and practised. When he died on July 25, 1936, the *British Medical Journal* described him as "a far-seeing and munificent friend" of medicine, and *The Lancet* referred to him as "a great inventor and innovator and an open-handed supporter of scientific progress". It is fitting that the medical profession of Australia should honour his memory at this time, and that his life-story should be briefly retold.

After education in a number of frontier schools, including the typical log-cabin school of the period, the lad went to work in a drug store owned by his father. It may be that early admiration for an uncle who was a prominent frontier surgeon turned his mind towards medicines and therapeutic remedies, for he showed an immediate interest in pharmacy and chemistry. After a year or two he left home and studied at Chicago and Philadelphia; he took his diploma at the Philadelphia College of Pharmacy when he was twenty years of age. As a pharmacist he was employed by two of the leading wholesale druggists in New York and travelled extensively in the United States, in Central America and in South America. We can imagine how his lively and inquiring mind was stimulated. He sent contributions to scientific publications and these gained recognition for him by leading pharmacists. He seems to have had a gift for friendship in the social and business worlds and we cannot doubt that he showed some business acumen. His journeyings took him into unusual places—he visited the cinchona forests of Ecuador and Peru and became interested in the production of quinine. Travelling by mule, he lost no opportunity of collecting information about primitive remedies used by the Indian tribes of South America. It must have been at this time that he began to develop the collector's outlook and habit which were to be such characteristic features of his later life.

Henry Wellcome was soon to start in business, and it was then that he told his mother that his life's ambition was "to amass great wealth and to devote it to the service of humanity"—an ambition which we see realized today. With his friend, Silas Mainville Burroughs, he left the United States for Great Britain, there to found in 1880 the firm which today bears the names of both friends. This is not the place to give a history of that firm, but in order to show what Henry Wellcome achieved it must be pointed out that the firm has extended its activities to many countries, having branches in Sydney, New York, Montreal, Cape Town, Milan, Shanghai, Bombay and Buenos Aires. When Silas Burroughs died in 1895 Henry Wellcome was left to carry on the business alone and he pursued the objective that he had stated to his mother. A number of research institutions and museums were established. In 1894 came the Wellcome Physiological Research Laboratories ultimately placed at Beckenham and in 1896 there followed the Wellcome Chemical Research Laboratories, established in London. Research was directed not only into the technical problems of manufacture and chemical control, but also into the investigation and development of new remedies. The Physiological Research and the Chemical Research Laboratories were eventually combined and became known as the Wellcome Research Laboratories. Wellcome for many years collected relics, books and data in connexion with primitive customs and ancient methods of medical and surgical treatment. He visited bazaars, old pharmacies, bookshops, dealers in antiques and owners of private collections. The Wellcome Historical Medical Museum was opened in 1913 in London at the time of the International Medical Congress. To quote the *British Medical Journal*: "This museum, the most extensive of its kind in the world, contains a permanent collection of exhibits illustrating the development of medicine, chemistry and the allied sciences from primeval times." The Wellcome Medical Historical Library contains Wellcome's collection of some 300,000 medical books and manuscripts, some of them dating back to the Middle Ages. One fact is certain—no Australian medical graduate should look on a visit to England as complete unless he has visited the Wellcome Research Institution in Euston Road, London. Wellcome went further afield. In 1900 he founded the Wellcome Tropical Research Laboratories at the Gordon Memorial Hospital, Khartoum, to which was added a floating research laboratory on the upper part of the Nile. He was interested in archæology and ethnology and he carried out explorations in the Upper Nile region of the Anglo-Egyptian Sudan. These activities were interrupted by the outbreak of the First World War. Wellcome had become a naturalized British subject by this time and he threw himself and his resources whole-heartedly into the cause of the Allies. He investigated the matter of army field ambulances and he presented to the Army Medical Services a motor field research laboratory which was used in Egypt and Palestine. During the South African War he had borne the entire cost of the medical and surgical equipment of the Hospital Ship *Maine*. In China he founded a fund to provide cheap standard medical, surgical and chemical text-books for the use of Chinese students. The Wellcome Medical Dispensary and the Lady Stanley Maternity Hospital in Uganda which were under the control of the

Church Missionary Society owed their existence to him. He supported an archæological expedition to the Near East and this discovered some valuable Biblical information. He also established scholarships and prizes. When Henry Wellcome died at the age of eighty-two years he left an estate the gross value of which was £2,138,959. His entire interests in the Wellcome Foundation were left in the hands of five trustees whose aims are defined in the will in the following terms:

> The advancement of research work bearing upon medicine, surgery, chemistry, physiology, bacteriology, therapeutics, materia medica, pharmacy and allied subjects, and any subject or subjects which have or at any time may develop an importance from the invention and improvements of medicinal agents and methods for the prevention and cure of disorders and the control or extermination of insect and other pests which afflict human beings and plant life in tropical and other regions and elsewhere.

It is small wonder that a man who did such great things for medicine, science and medical history should be honoured by learned societies, universities and governments. Honours were heaped upon him. In 1927 Henry Wellcome was given the honorary degree of Doctor of Laws by the University of Edinburgh. (The British Medical Association was holding its annual meeting at Edinburgh at that time.) The Royal College of Surgeons of England made him an honorary Fellow in 1931. In 1932 he received the honour of knighthood and was elected a Fellow of the Royal Society. He was an honorary Freeman of the Society of Apothecaries. He was given the honorary degree of Doctor of Science at the Marquette University, Wisconsin. He received the cross of Officer of the Legion of Honour of France and was created *Comendador de la orden de la República* in recognition of his outstanding services to Spanish interests.

When we reflect upon this man, who was not a medical graduate, but who laboured abundantly in the cause of medicine and medical science, leaving behind him a world-wide organization which devotes the whole of its profits to research, we do well to ponder and to ask ourselves whether each of us, in his or her own small sphere, should not try to give back to medicine in endowment, large or small, something of what it has given to us.

COMMONWEALTH BURSARIES.

For many years this journal, in discussions on the granting to post-graduate students of overseas Dominions and other parts of the British Commonwealth of fellowships, bursaries and other awards instituted to further research in medicine and the allied sciences, has pleaded for a two-way traffic between the Mother Country and other parts of the Commonwealth. Our contentions have dealt not only with new graduates and graduates of an older vintage, but also with those who teach. It was with the greatest satisfaction that Australians greeted the recent appointments of Professor H. R. Dew, of Sydney, as Sims Travelling Professor and of Professor B. T. Mayes, also of Sydney, as Sims-Black Professor of the Royal College of Obstetricians and Gynæcologists for 1954. If the British Commonwealth is to be real and alive and to fulfil its true destiny, any wealth which its several parts have in common must be based on mutual understanding of national problems, of national difficulties and of national traditions. On this foundation alone can a sound superstructure be built. Once the foundation of a structure has been laid the building above may have many uses and a varying composition. In our Commonwealth structure there can be no feature more necessary, more effective, more durable and more catholic in its appeal than science. For this reason all scientists, medical and non-medical, will welcome the announcement that a series of Commonwealth bursaries are to be established by the Royal Society in England and the Nuffield Foundation.

This announcement has been made simultaneously in the *British Medical Journal* and *The Lancet* of August 1, 1953. The object of the bursaries is to increase the efficiency of investigators who have proved their worth by enabling them to pursue research, to learn techniques and to follow other forms of study. As a rule bursaries are awarded to a worker to enable him to attack and possibly solve some particular problem; but the object of these bursaries is quite different, for it aims at improving the powers of the recipient to extend the bounds of knowledge. For the first five years the scheme will be on an experimental basis. The funds will be provided by the Nuffield Foundation to the extent of £5000 a year. Bursaries will be awarded to scientists in the United Kingdom who wish to go overseas to different parts of the Commonwealth, and to scientists of one oversea part of the Commonwealth to go to another. For a start the Royal Society will also make a contribution of £2500, which will be available to a worker travelling in any direction within the Commonwealth. We read also that it is hoped that funds may be obtained from other sources, particularly from overseas, so that more bursaries will become available for scientists of oversea countries of the Commonwealth who wish to study in the United Kingdom. *The Lancet* prints the following quotation:

> Each bursary will provide for the cost of travel and maintenance normally for periods of two to twelve months. It will not aim at the provision of any salary as such, but of sufficient maintenance to avoid frustration. The applicant must be sponsored by a recognized research authority and must have prior permission to work in the laboratory or other scientific institution chosen.

A joint Commonwealth Bursaries Committee has been set up. The Chairman of this committee is Sir Edward Salisbury, the Secretary of the Royal Society. Three members are to be nominated by the Trustees of the Nuffield Foundation, three have been nominated by the Council of the Royal Society. The Nuffield Foundation group comprises Mr. Leslie Farrer-Brown, Sir John Stopford and Professor A. R. Todd; the Royal Society group comprises Professor G. R. Cameron, Professor H. S. W. Massey and Sir Eric Rideal. Bursaries will be awarded twice a year, and applications will be submitted not later than March 15 and September 15 of each year. The first applications will be dealt with in 1954.

Another recent happening may be noted here, though it does not come under the heading of Commonwealth Bursaries. An Australian practitioner has been appointed temporary lecturer of child health in the University of Liverpool. Some little time ago Professor Norman B. Capon, the professor of child health of Liverpool, wrote to Professor Lorimer Dods, of Sydney, stating that his lecturer was to spend six months in the United States, and advising him that a vacancy was about to occur on

the staff of the Liverpool department. Thereupon Professor Dods discussed the matter with Dr. Donald G. Hamilton, of Sydney, and Dr. Hamilton applied for the position. He has received the appointment, and is shortly to travel to England by way of the United States. This shows that Professor Capon at least thought that there might be overseas pædiatricians capable of occupying the position. There is no doubt that this kind of temporary exchange between universities in the Commonwealth, this two-way traffic, will be of great benefit to Commonwealth medicine.

Current Comment.

THE MEDICAL ADVENTURES OF SIR WALTER SCOTT.

Most people in countries where English is the spoken language are familiar with at least the novels of Sir Walter Scott. In the third edition of "Chambers's Cyclopædia of English Literature", the following comment is made on his literary importance:

> That long array of immortal fictions [the Waverley Novels] can only be compared with the dramas of Shakespeare, as presenting a vast variety of original characters, scenes, historical situations, and adventures. They are marked by the same universal and genial sympathies, allied to every form of humanity, and free from all selfish egotism or moral obliquity. In painting historical personages or events, these two great masters evinced a kindred taste, and not dissimilar powers. The highest intellectual traits and imagination of Shakespeare were, it is true, not approached by Scott: the dramatist looked inwardly upon man and nature with a more profound and searching philosophy. He could effect more with his five acts than Scott with his three volumes. The novelist only pictured to the eye what his great prototype stamped on the heart and feelings. Yet both were great moral teachers, without seeming to teach. They were brothers in character and in genius, and they poured out their imaginative treasures with a calm easy strength and conscious mastery, of which the world has seen no other examples.

Scott also achieved fame as a poet and as a biographer, but at the moment we are more concerned with his chequered medical career than with his literary activities.

An interesting study is presented by Esther H. Vincent, entitled "Scott of Abbotsford".[1] She describes how, when Walter Scott was born in the ancient city of Edinburgh, on August 15, 1771, his birthplace was "just awakening from the dank placidity of its unhygienic past". His father was a Writer to the Signet. His mother, Anne Rutherford, was the daughter of a physician who was professor of medicine in the University of Edinburgh, and had studied abroad, notably in Leyden with Boerhaave. The first six children of Walter and Anne Scott all died in infancy. The second six, born after the family had moved from their previous rather slum-like dwelling, lived. Walter, junior, was the fourth of the surviving children. When he was eighteen months old he caught a fever which his elders thought might be due to teething. Some days later he was noticed to have lost the use of his right leg. His maternal grandfather could find no cause for the condition and other eminent physicians of the city could make no satisfactory diagnosis. In Esther Vincent's words, it was to be another fifty years before poliomyelitis was differentiated. The unfortunate child was subjected to all kinds of treatment, including blistering, a removal to his paternal grandfather's farm to take the virtues of the country air, and being wrapped up in the still warm skin of a freshly killed sheep. Thus attired, he was laid on the parlour floor, while Grandfather Scott tried to induce the small cripple to crawl. His health recovered, but not his lameness. However, his family did not give up hope, and at the age of four years he was taken to Bath, where he "stoically endured the water regimen of pump room and bath". He continued to limp. When he was eight years old, he took a course of sea-bathing at Prestonpans, which did him no more good than the sheepskins or the Bath waters. Attempts to expose him to formal education in Edinburgh were not eminently successful; he preferred border songs and legends to arithmetic. He was a sturdy child, but when he was twelve years old he seemed to be losing some of his strength, and so was sent back to Grandfather Scott's farm for a while. At the age of fourteen years he had a serious hæmorrhage, caused, so it was said, by the "bursting of a blood vessel in the lower bowel". The treatment which he received is so remarkable that it is worth quoting.

> Although the Edinburgh weather was cold and raw that spring, he was covered with but a single blanket. He was bled and blistered until he plaintively said that the remedy was worse than the illness. Talking was prohibited and his diet was reduced to vegetables. His uncle, Dr. Daniel Rutherford, attended him, and thought his recovery a miracle, as indeed it must have been, considering the treatment.

Esther Vincent thinks that it is possible that he had ulcerative colitis, although that condition is rare at the age of fourteen years. During his long convalescence, he read his favourite romantic tales, played chess, and by a special arrangement of mirrors, watched the soldiers marching by.

Scott grew into a tall and muscular young man, with a high, almost conical head. Craniometrically, although not clinically, he was megacephalic, and some medical men think that his oddly shaped skull is due to the fact that while he was growing, his head was deprived of the upright position for long periods during his illnesses. In 1797, while visiting the English Lakes, he made the acquaintance of Charlotte Mary Charpentier (or Carpenter), whom he eventually married. They had four children, two sons and two daughters. Until the winter of 1816-1817 he enjoyed robust health and played and worked with great vigour. The trivial ailments that fell to his lot he treated in Spartan fashion. Esther Vincent tells us that from youth to age he was reluctant to resort to physicians or drugs. Then, for more than two years, he suffered intermittently from violent attacks of biliary colic. This was hereditary in his family, but his dietary indiscretions may have contributed to his downfall, although he was only a moderate drinker of alcohol. The attacks reached their climax in the spring of 1819, but during this period he never seems to have considered surgical treatment, although he nearly died. He described the onset of acute cholecystitis as "cramp setting fire to its lodging". During an exhausting ten-day attack he was alone at Abbotsford with his elder daughter Sophia, and he seems to have suffered as much from the medical treatment he received as from the disease. He was bled and blistered and given ipecacuanha and calomel, laudanum and opium, but he still had unrelieved pain. After a night of agony he would get up early in the morning and ride twenty miles on his horse. These repeated attacks caused his hair to turn white and his skin to turn yellow, and he became emaciated. One day, when he had an attack, he took a large dose of calomel, and when the pain almost immediately departed he thought he had cured himself, especially since he never again had a severe attack. It is noteworthy that during these two years of misery he wrote the whole of "The Bride of Lammermoor" and parts of "Ivanhoe" and of "Rob Roy", but he could never remember what he had written. He always said that "Rob Roy" smelled of cramp.

In 1820 he received a baronetcy at the hands of George IV, and in the same year was made president of the Royal Society of Scotland, although he knew little of science. In 1821 he attended the coronation of George IV, and in 1822 was master of ceremonies at the King's visit to Edinburgh. He afterwards developed a curious skin disease, "a very inconvenient and nasty eruption", which remained on his legs and arms for years. In December, 1825, he had a severe attack of renal colic with violent pain in the right kidney. Fortunately, this was not repeated. About the same time he had some kind of urinary disorder, the

[1] *Surg., Gynec. & Obst.*, May, 1953.

exact nature of which it is impossible to discover. However, he himself was obviously alarmed and felt sure that he would die within a few years. "Square the odds, and goodnight, Sir Walter, about 60." Scott had all his life been subject to intermittent attacks of a nervous disorder, which he sometimes described as "*morbus eruditorum*" and sometimes as the "black dog". He wrote in his journal that it was characterized by heart tremor, baseless fear and lassitude. This type of neurosis is not entirely unfamiliar at the present time. Several attacks of severe "rheumatism" in the winter of 1826-1827 prevented him from exercising, and he himself thought that the final breakdown of his health was due to this. To add to his troubles, in 1826 his publishers went bankrupt and with their bankruptcy vanished his fortune. He had saved no money, having spent his substance on land and hospitality and on helping others, and his only hope of repaying his creditors was by a "crushing schedule" of writing. In May, 1826, also his wife fell ill and later died. Of her illness he wrote as follows:

> The complaint is of water in her chest, and the remedy is a foxglove, which seems a cure rather worse than most diseases, yet she sustains both disease and the remedy to the surprise of medical persons.

Scott's father, mother and brother had all died of apoplexy, and it is likely that there may have been an hereditary tendency to arteriosclerosis in his family. The first hint that he may have been affected was given in 1822. In June, 1829, he had a vague attack which passed off. He was treated by "copious cupping". His first definite cerebral catastrophe occurred on February 15, 1830. In this attack he became unable to speak and remained so until a surgeon came in about ten minutes and bled him. This attack frightened him so much that he gave up smoking for a time. He refused to have a medical student as his secretary, and so his surgeon, Mr. James Clarkson, privately instructed a servant in the use of the lancet. Scott had another, but slighter, stroke in November of the same year, and he now realized what his illness was. He was not afraid of death, but he was afraid of mental deterioration. However, he faced his probable future bravely. Another more severe attack in April, 1831, left him with an obvious distortion of the cheek muscles, some hesitation in speech and signs of mental impairment. Under these conditions he tried to take part in the May electioneering. By July he was well enough to take a short driving tour with his son-in-law, and in September he went onto the Continent to seek further improvement in his health. He was now under the illusion that he had paid all his debts and could stop driving himself with his writing. In London he was examined by Dr. Robert Ferguson, Sir Henry Halford and Dr. Holland. All three concurred in a diagnosis of incipient brain disease and suggested that if he ceased working, the disease process might be arrested. Scott visited Malta, Naples and Rome, and on the way back to Britain via the Rhine, he had another attack, relieved by his servant's lancet. On June 13 he was again examined by his three doctors in London, but by this time he was in a kind of waking dream, although he still retained his personal dignity. He returned to Abbotsford, where, with only one interval of consciousness, he lingered on for two months. He died on September 21, 1831. An autopsy, limited to his head, was performed by his physician, Mr. Clarkson, in the presence of Dr. Adolphus Ross, of Edinburgh. The report is given by Esther Vincent as follows:

> On removing the upper part of the cranium, the vessels on the surface of the brain appeared slightly turgid, and on cutting into the brain the cineritious substance was found of a darker hue than natural and a greater than usual quantity of serum in the ventricles. Excepting these appearances, the right hemisphere seemed in a healthy state, but in the left, in the choroid plexus, three distinct though small hydatids were found; and on reaching the corpus striatum it was discovered diseased—a considerable portion of it being in a state of ramollissement. The blood vessels were in a healthy state. The brain was not large—and the cranium thinner than it is usually found to be.

No attempt has been made in this short account to discuss Scott as a literary figure, or his legal and military interests. We have been concerned only with the maladies that beset him and with the remedies that were inflicted upon him. We cannot but admire the courage with which he faced his fate and the amount of work that he accomplished, nor can we wonder that he had no particularly warm regard for the medical profession, although he did express his gratitude to and admiration of Clarkson; but his summing up of the medical profession occasions no surprise:

> A doctor is like Ajax—give him light and he may make battle with a disease; but, no disparagement to the Esculapian art, they are bad guessers.

ANKYLOSING SPONDYLITIS IN WOMEN.

In Comroe's standard text-book "Arthritis and Allied Conditions" Boland has written the section on ankylosing spondylitis under the title "Rheumatoid (Ankylosing) Spondylitis" and thus reflects the American opinion on its ætiology. English physicians, on the other hand, tend to regard it as ætiologically distinct from rheumatoid arthritis, and one of their most telling arguments is the difference in sex incidence, for while it is well known that rheumatoid arthritis occurs four or five times as frequently in the female, it is equally clear that ankylosing spondylitis is just as predominant in the male. R. Mowbray, A. L. Latner and J. H. Middlemiss reported a series of 19 women and 118 men;[1] in C. W. Buckley's series[2] there were six women and 54 men.

T. L. Tyson, W. A. L. Thompson and C. Ragan,[3] obviously troubled by this weakness in the concept of unity of the two diseases, have reviewed their case histories of the past twenty years in search of an explanation, stating:

> It occurred to us that there might be differences in the clinical manifestations and course of the disease which made the difference in sex distribution more apparent than real.

In twenty years they had studied 60 cases in women and over 450 in men. It had been their impression that the disease was milder in women, and therefore fewer found their way past the general practitioner to the specialized clinics from which the series such as those above had been reported. In almost all clinical features, however, there was little difference from the clinical picture in males. The age of onset varied from fourteen to fifty-nine with a mean of twenty-five years. The neck region was involved in 41 (68%), a much higher incidence than in male patients, but the distribution in other parts of the spine was much the same as in males. Radiological evidence of involvement of the sacro-iliac joints was found in 56 (96%) and involvement of the *symphysis pubis*, rarely seen in males, was found in six (10%). Anæmia was present in 30% and the sedimentation rate was raised in 86%. Marked systemic manifestations such as anorexia and weight loss, pallor, exhaustion and tendency to deformity were usually lacking in women and only seven showed a degree of severity comparable to that seen in the male. Tyson and his co-workers concluded, however, that the differences in the two sexes are not great enough to have caused a fallacy in the reported sex incidence. In six (10%) of their cases there was a definite family history; and a high familial incidence in women has been noted by others. In a paper read to the Medical Research Society of London in February, 1949, West of Bristol reported that four of his fourteen women patients had siblings affected compared with six of sixty-nine males. As there appeared to be some barrier to development of the disease in the female, he suggested that in those women who did suffer, the penetrance of the affected gene must be great and thus the family history prominent.

Management of the female patient raises special problems. It is interesting that two of the patients in the series of Tyson *et alii* had successful pregnancies and symptoms became less pronounced during the pregnancy.

[1] *Quart. J. Med.* (1949), 18: 187.
[2] *Brit. M. J.* (1931), 1: 1108.
[3] *Ann. Rheumat. Dis.* (1953), 12: 40.

A third patient developed jaundice during her pregnancy and miscarried, the spondylitis decreased and she remained symptomless during the next five years. In the majority of patients, both men and women, deep X-ray therapy causes lasting relief from pain and stiffness. In women, however, effective therapy in the lumbar region is very close to a sterilizing dose, and if the patient is in the child-bearing age, therapy must be directed obliquely and with effective screening in order to protect the ovaries.

Though it provides no explanation for the different sex incidence, the paper by Tyson, Thompson and Ragan is a timely reminder that mild and vague symptoms in the lower part of the back, in women, so often attributed to neurosis, or to disease or displacement of the pelvic organs, may in fact be due to ankylosing spondylitis, and radiological examination of the sacro-iliac joints may save the patient from years of frustration, endless phenobarbitone administration and unnecessary gynæcological operations.

IMMUNOLOGICAL MECHANISMS IN CERTAIN BLOOD DISORDERS.

THE interest in those immunological mechanisms which may invoke a cause for some of the acute and serious blood disorders has been sharpened in the last few years by the observed effects of ACTH and cortisone in these conditions. Leucopenic crises are a case in point. A number of years ago the so-called agranulocytic emergencies were thought in some instances to be due to an allergic cause, and the well-known relationship between sensitivity to and severe toxic reactions from certain drugs, such as amidopyrin, then received a good deal of attention in current literature. Hæmolytic anæmia has also been carefully studied from the same point of view. It may be remarked, for instance, that cases have been recently published in which the occurrence of hæmolytic anæmia has been noted a considerable time after exposure to known irritants which are believed to bear a causal relationship to the disease. Kai Bent Hansen has published a case report of the occurrence of agranulocytosis in a woman, aged sixty-three years, who had suffered from rheumatoid arthritis for which no drug treatment had been given.[1] She had a history of several recent febrile attacks, and was found to have only 1% neutrophile and 2% eosinophile cells in the blood, while bone marrow examination showed a profound depression of the myeloid series from the segmented neutrophile back to the promyelocyte. No definite cause could be assigned for the emergency, and it was decided to try the effect of ACTH given with a diet having a low salt and potash content. The eosinophile cells disappeared from the blood, as is common after the administration of ACTH, and neutrophile cells rose in number to 70% and 80%, with a corresponding fall in lymphocytes. With these changes the γ globulin content was observed to fall, though a rise occurred again when the treatment was temporarily suspended on account of sodium retention. The treatment was then continued, and after over a month the condition of the patient became stabilized, and the γ globulin remained at a normal level, while a satisfactory number of granular cells was constantly found in the blood. The author noted some similarity to the general picture of acquired hæmolytic anæmia, and on reviewing the investigations made during the whole of the illness, suggests that this patient, one of the so-called idiopathic group, had some abnormality of the antigen-antibody response of the body. This was not related to her arthritic condition, which was inactive, and was accompanied by a normal antistreptolysin titre. Hansen finally offers the ætiological concept that the disease might be a leucocytic variant of hæmolytic anæmia of the immunological type.

The subject of the existence of an immuno-vascular blood disorder is also dealt with by William Damashek in an editorial published earlier this year.[2] He draws a clear clinical picture of what he calls "acute vascular purpura". A patient previously well is found suddenly to have a very severe generalized purpura, often involving the mucous membranes. The platelets are very few in number, and marrow examination shows corresponding changes. The history in such cases usually includes an acute though brief infectious disease, or the administration of a drug previously given intermittently. In addition, injury to small blood vessels is commonly found; this may well be of importance in causing bleeding, for bleeding and purpura often subside before the number of blood platelets is restored to normal. Damashek points out that this condition resembles the other types of purpura in which thrombocytopenia is not observed. He quotes the work of Katsura, who produced endothelial antibodies in animals, and by injection into other animals, was able to reproduce generalized purpura. Other workers have confirmed these results. These findings are consistent with an immunological ætiology of acute vascular purpura, which in turn is strongly suggested by the effects of ACTH and cortisone in these states. There is also reason to think that *periarteritis nodosa* is a pathological state of the same order. Damashek suggests that disseminated lupus may belong to the same group, and finally sees the possibility that the general term "immunohæmatology" may win a place in terminology and medical thought akin to that now occupied by the adaptation syndrome of Selye.

[1] *Acta med. scandinav.*, 1953, Volume CXLV, Number 3.
[2] *Blood*, April, 1953.

ANTIBIOTICS: OUR PRESENT WEALTH.

EVER since antibiotics came into general use the voluminous literature on them has been liberally seasoned with articles advising caution. Pathologists and pharmacologists have explained basic principles, clinicians have described side-effects and reported cases, public health officers have asked for economy, medical editors have pleaded, reasoned and fulminated—all in an effort to have antibiotics used reasonably. Probably no major therapeutic agent yet discovered has come nearer the ideal than this group of substances. Relative to their great therapeutic power the immediate disadvantages associated with their use are normally small. Clinicians are familiar with and prepared for such side-effects as allergy to penicillin and toxic reactions to streptomycin; the development of blood dyscrasias, reported overseas and attributed to chloramphenicol, has not been common. Superinfection is a larger problem and requires more emphasis, particularly in the light of a recent report by C. Brown, junior, *et alii*[1] of five fatal cases of fungous infections complicating "broad spectrum" antibiotic therapy. However, we do not wish to spend time at the moment on the side-effects of antibiotics on the patient. These were considered in the issue of June 28, 1952, in a paper by F. H. Hales Wilson and in a leading article entitled "Antibiotics Amok". None of the side-effects has outlawed a particular antibiotic after it has come into general use, other than dihydrostreptomycin, although they emphasize the need to use any powerful drug only in accordance with its strict indications and then only if really necessary.

The side of the question that we wish to emphasize, because it is much more subtle and at the same time much more dangerous in the ultimate, is the appearance of antibiotic-resistant organisms. Edgar Thomson's paper in the issue of June 28, 1952, provided facts and figures that should make any responsible doctor pause every time he thinks of prescribing or administering an antibiotic. In this issue we publish a letter from Dr. Thomson, again pleading for moderation and intelligence in the use of antibiotics. We would commend it for careful reading, together with the table appearing under the heading of "Medical Practice". Especially important is the advice that the newer antibiotics be kept strictly in reserve. There seems a real danger that if we do not learn to value the therapeutic wealth that lies in the antibiotics, it may become like the fairy gold in the old tales, which turned to withered leaves in the hands of those who misused it.

[1] *J.A.M.A.*, May 16, 1953.

Abstracts from Medical Literature.

DERMATOLOGY.

Treatment of Perungual Verrucæ.

L. FRANK (*Arch. Dermat. & Syph.*, May, 1952) describes a treatment of perungual verrucæ. He first applied 90% strength trichloracetic acid to the verruca; after this he applied liquid phenol, and then occluded the verruca completely with an adhesive dressing. He found that when the adhesive was removed after one week, the wart was dried and adhered to the adhesive, and the entire wart came off with the adhesive dressing, leaving a clean bed underneath. Occasionally, when the wart did not come off with the adhesive, simple curettage was sufficient to remove the remaining dried and crumbly material which was all that remained. Patients frequently complained of pain on the second or third day after the treatment, but in only two cases was the pain severe enough to require removal of the occlusive dressing.

Diethylstilbœstrol and Senile Sebaceous Adenoma.

W. C. LOBITZ AND D. P. COLE (*Arch. Dermat. & Syph.*, September, 1952) state that basic evidence is increasing to indicate that the androgens and the œstrogens, when given internally or applied topically, influence the size of the sebaceous gland in laboratory animals as well as in human beings. Androgens (for example, testosterone propionate) cause an increase in the size of the sebaceous gland. On the other hand, œstrogens (for example, diethylstilbœstrol) effect a decrease in the size and number of the sebaceous glands in humans and laboratory animals. The authors observed the clinical effect of systemic œstrogen therapy upon the size and activity of sebaceous glands in a patient with multiple tumours of senile sebaceous adenoma. The patient, a sixty-two years old man, had multiple cystic and tumour-like lesions of the face. Cheesy material could be expressed from some of the lesions. From the age of fifty-five years his pillow slip each morning was stained with a yellow oil from his face. On examination of the patient, he was found to have a very oily skin, dilated sebaceous gland openings, umbilicated on their surfaces, mild erythema and telangiectasia, all present on the face, neck, upper part of the chest and back. The only treatment given was diethylstilbœstrol by mouth. The seborrhœa did not lessen with small amounts of œstrogen. It seemed that not until breast hyperplasia had begun did the sebaceous element show dramatic response. The dosage used was from one milligramme daily up to five milligrammes daily.

Pyogenic Infections of the Skin.

J. W. JORDON (*New York State J. Med.*, September, 1952), in treating the pyodermas of 243 patients, used the following antibiotics given topically, parenterally or orally, singly or in conjunction: aureomycin, bacitracin, terramycin, "Chloromycetin", neomycin, aerosporin and penicillin. The antibiotics used topically were employed in ointments, aqueous solutions, shake lotions and occasionally other media. Sulphonamides, penicillin, streptomycin and dihydrostreptomycin were not employed as topical applications because of their well-known sensitizing properties. Penicillin, however, was frequently employed, either orally or parenterally, when special indications made this advisable. Stable ointment may be made from aureomycin, bacitracin, terramycin, "Chloromycetin", neomycin and aerosporin. Aureomycin and bacitracin deteriorate rather quickly in an aqueous solution. "Chloromycetin", neomycin and aerosporin are soluble in sufficient concentration for therapeutic purposes and are stable enough for practical considerations. Aureomycin, terramycin and "Chloromycetin" can be used not only topically but also systemically. All six antibiotics used as topical applications showed a low incidence of cutaneous sensitivity. The author concludes that simple superficial pyogenic infections of the skin usually respond to topical treatment with one or more antibiotics. Topical antibiotic treatment is an aid in the management of *sycosis vulgaris* and external otitis, but the percentage of complete cures of these conditions is much lower than in simple infections. Routine cultures or sensitivity tests are not usually necessary as guides in the antibiotic treatment of simple pyodermas. They may be useful in refractory cases or for unusual clinical conditions. Neomycin appears to be the best antibiotic for topical treatment of the superficial pyodermas because of its wide bactericidal spectrum, its stability in aqueous solutions and ointment bases, and its low sensitizing index, and because it is seldom used systemically.

Secondary Eruptive Xanthomatosis due to Myxœdema.

A. C. CURTIS AND H. C. BLAYLOCK (*Arch. Dermat. & Syph.*, October, 1952) report a case of hypercholesterœmia, myxœdema and secondary eruptive xanthomatosis which compares clinically with three previously reported cases. They state that xanthomatous lesions, clinical myxœdema, a low basal metabolic rate and high blood cholesterol and high cholesterol ester values are apparently characteristic of this entity. Involution of the xanthomatous lesions and lessening of the myxœdema occurred during thyroid medication.

Calcium Ointment and Atopic Eczema.

T. N. GRAHAM (*New York State J. Med.*, July 1, 1952) states that it is well known that multiple allergies are an important factor in the causation of atopic eczema. Therapy, including topical applications, has resulted in very little benefit. Allergy tests in this disease have failed to be of value. Psychogenic factors should be investigated in all cases. A review of the literature shows that Tobias found 10% calcium ointment of definite value in selected cases of subacute and chronic types of atopic dermatitis. In a chemical study of atopic eczema, Engman and MacArdle called attention to the consistent diminution in the amount of the white ash of calcium and magnesium in the entire epidermis of patients with atopic dermatitis (generalized neurodermatitis) compared with epidermis from both normal subjects and patients with a variety of cutaneous diseases. A survey of foreign literature reveals that Kunzle reported that hypersensitivity of the skin depended on the ion equilibrium. The author treated 22 persons suffering from chronic atopic dermatitis with 10% calcium gluconate in an ointment base made of organic substances dispersible in water. These patients had previously had other local therapy, some of them X-ray therapy, without improvement. The results were as follows: no improvement, nine cases; improvement, five cases; definite improvement, eight cases. In all cases studied, a placebo ointment was used on part of the epithelium in order to check the results. Three case reports are given as representative of the cases in which response was favourable to calcium ointment therapy.

Essential Hyperidrosis and Hexamethonium Bromide.

J. SOMMERVILLE AND J. B. MACMILLAN (*Brit. J. Dermat.*, December, 1952) state that Burt and Graham, when testing pentamethonium and hexamethonium in cases of hypertension and peripheral vascular disease, noted that sweating was reduced in normal subjects, and in a case of hyperidrosis the excess sweating was rapidly controlled. Tolerance developed rapidly in their case, and treatment was discontinued. Prompted by the currently accepted concept of nervous control of sweating and by the isolated finding of Burt and Graham, a trial was made in treating subjects of localized (essential) hyperidrosis with hexamethonium bromide. Six patients were given hexamethonium bromide by mouth. These had moderately severe or severe examples of the condition, and had resisted previously attempted control with astringents, sedation and atropine. All were admitted to the wards, and after full investigation to exclude organic disease, hexamethonium bromide was given in 250 milligramme tablets and the dose gradually increased to 1250 milligrammes daily, spread over the waking hours. Baseline blood pressure readings with the patient in bed and ambulant were made prior to treatment, and these were continued after commencement of treatment. In only one case was there any significant fall in blood pressure. No patient had visual or salivary disturbance. In all six cases the hyperidrosis was controlled initially, and the minimum period of control was two months. The effect, although considerable, was not as complete in the axillæ as on the palms and soles. In two cases the condition was controlled for periods of three and four months respectively by administration of 1250 milligrammes daily, and has remained controlled for five months since administration of the drug was stopped.

Mixed Tumour of the Lip.

E. P. CAWLEY AND C. E. WHEELER (*Arch. Dermatol. & Syph.*, September, 1952) consider that mixed tumours of the lip may be commoner than the paucity of reported cases indicates. The parotid and submaxillary glands are the commonest locations of mixed tumours of salivary gland type. In addition to those in the salivary glands, mixed tumours of this type have been encountered in the hard and soft palates, lachrymal glands, lips, tongue, buccal mucosa, pharynx, nasal cavities and floor of the mouth. The mixed tumour of the lip is usually round or

oval, and both the overlying skin and the mucous membranes are intact. On palpation, the lesion is painless, circumscribed, movable and usually firm, but it may, in some instances, give the impression of being cystic. Mixed tumour of the lip must be distinguished from the common mucous retention cyst of the lip. Mixed tumours of the salivary gland type may vary widely in histological appearance, but they have two essential features in common wherever they occur: (*a*) the presence of epithelial elements, (*b*) the presence of mesoblastic elements. The epithelial elements may include glandular epithelium, which is almost always present, and squamous epithelium, which is noted in the majority of cases. Mesoblastic elements may include cartilage, mucoid tissue, connective tissue and, rarely, adipose, muscle and lymphoid tissue. Mixed tumours of the lip are almost invariably benign. They are best treated by surgical excision, and do not recur if completely removed.

UROLOGY.

Hyperchloræmic Acidosis of Uretero-Sigmoidostomy.

J. D. MITCHELL AND W. L. VALK (*J. Urol.*, January, 1953) conclude from their studies that all humans reabsorb chloride, sodium and ammonia from the rectum in a fairly uniform manner. All patients with urinary diversion to the sigmoid colon have the potentiality of developing hyperchloræmic acidosis. This development does not depend on the level of renal function or abnormal distal tubular function. The blood levels of non-protein nitrogen and urea nitrogen are raised because of reabsorption of ammonia and its conversion to urea by the liver, and not because of reabsorption of urea from the rectal urine. Proper management of patients with uretero-colic anastomosis must include (*a*) a low chloride intake, (*b*) a large fluid intake, (*c*) frequent emptying of the rectum and (*d*) supplementary sodium intake, usually as one to four grammes of sodium bicarbonate per day.

Cushing's Syndrome from Adrenal Cortical Adenoma.

G. D. OPPENHEIMER *et alii* (*J. Urol.*, September, 1952) state that a case of surgical cure of Cushing's syndrome due to adrenal cortical adenoma is sufficiently rare to warrant report. They can find only three other cases in the literature. The patient was a single woman, aged twenty-six years, who had been troubled for two years by severe attacks of pain in the ribs and the lumbar region, by amenorrhœa, though before that the menses had been normal, and by development of a "moon facies" and localized trunk adiposity. She had facial hirsutism, and many points of tenderness over the ribs. The glucose tolerance curve was diabetic in character. Urinary excretion of the neutral 17-ketosteroids was within normal range. Fifty-three fractures of the ribs and sternum were discerned, as well as compression fractures of all the lumbar and some of the thoracic vertebræ. No abnormality was noted after bilateral perirenal insufflation in conjunction with excretion urography, even with tomographic technique. In view of the absence of a demonstrable adrenal tumour by X-ray examination, and since the *sella turcica* was radiologically normal, a diagnosis of bilateral adrenal cortical hyperplasia was made. Androgenic therapy was instituted in an effort to increase the deposition of protein matrix in the skeletal system, and thus aid in the recalcification of the bones. This was a complete failure, since the disease progressed. Then left lumbar adrenal exploration was carried out, the left side being chosen arbitrarily. No tumour was found, but, on the contrary, a very atrophic adrenal gland. This was left alone, as it was realized that it probably reflected the presence of a functioning adrenal tumour on the opposite side. Three months later, after preparation with whole and lipo-adrenal cortical extract, the right suprarenal area was explored. Here was seen a smooth, encapsulated, yellowish-brown tumour, three centimetres in diameter, and weighing nearly ten grammes. It was removed, a very small fragment of atrophied adrenal tissue being left behind. Microscopically the neoplasm consisted of fairly uniform cells in clusters separated by capillaries. No cell atypism or mitosis was seen. The patient was fairly weak for twelve hours, when a precipitous fall of blood pressure occurred, with the clinical picture of severe shock. With vigorous treatment by whole and lipo-adrenal cortical extract and other measures, she climbed out of this desperate condition after thirty-six hours of danger, but was still in intermittent danger for some time from sudden episodes of shock. There were many other rather serious troubles, but she was finally discharged about six months after the adrenalectomy. Post-operative study at the end of sixteen months after the operation revealed regular menses and a striking change in facial appearance, with disappearance of the hirsutism and the trunk obesity. The basal metabolic rate, previously *minus* 34%, was now *plus* 2%.

Testicular Tumours.

C. RUSCHE (*J. Urol.*, July, 1952) has abstracted the medical records of 131 cases of testicular tumour. The patients, observed over six years between 1942 to 1948, all had a confirmed histological diagnosis. Only 7% of cases were benign. The overall survival rate in the series of malignant tumours was 54%. The author states that the survival rate may be correlated with the degree of differentiation of the neoplasm, since the five-year rate was for seminoma 61%, for embryonal carcinoma 66%, for adult teratoma 43%, and for terato-carcinoma 21%. When the period of survival is over two years the prognosis becomes more favourable, since extension of the disease after this time is generally confined to a solitary metastatic lesion. Malignant tumours of the testis are a local disease for a certain period of time. If the diagnosis is made, and orchidectomy performed within this period of time, hope of cure is excellent. In four of the fifteen cases associated with cryptorchidism the normal mate was the seat of the neoplasm. In four cases in which orchidopexy had been performed with no evidence of neoplasm at the time of operation, the patients later developed malignant tumours. Therefore, this operation does not necessarily protect the patient against the later development of a malignant neoplasm in the testicle concerned. The author states that the treatment of testicular tumours depends largely on their type. It is important to have serial sections examined from the removed testis, because of the variation in histological appearance which may occur in one specimen. The prognosis is generally that of the most dangerous part. Pre-operative irradiation should be condemned because (i) valuable time may be lost before the tumour is removed, (ii) irradiation alters the original architecture of the tumour, so complicating accurate microscopic diagnosis and therefore proper prognosis, (iii) benign tumours may inadvertently be irradiated, and (iv) the tumours vary in their radiosensitivity. The therapeutic value of post-operative irradiation has been shown in greatly increased survival rates, but some tumours are radioresistant. The adult type of teratoma does not respond to irradiation. A lethal dose of radiation for seminoma is only about 1500*r* to 2000*r*, an amount which can be delivered without injury to surrounding structures. If there is an admixture of other tumour types with the seminoma, irradiation should be in larger doses. Embryonal carcinoma and terato-carcinoma are much more resistant, and call for dosage varying from 5000*r* to 7000*r*. This frequently distresses the patient, and may cause late radiation injuries.

Terramycin in Urinary Tract Infections.

H. M. TRAFTON AND H. E. LIND (*J. Urol.*, February, 1953) state that, owing to problems like the development of bacterial resistance to antibiotics, it is better to make a full bacteriological study, including determination of sensitivities, so that one or more suitable antibiotics may be used from the outset. Refinement and standardization of the disk method of determining sensitivities have facilitated this matter in an inexpensive way. In this study 35 patients were selected because the majority had failed to respond to sulphonamides, streptomycin, aureomycin, penicillin or chloramphenicol. Terramycin was found to be tolerated better than aureomycin, about the same as chloramphenicol, but less than penicillin or streptomycin. Some degree of diarrhœa was present in about one-third of the cases, but it was not severe enough to interfere with treatment. Most of the patients received two capsules every six hours for six days, that is, two grammes per day. In the group with uncomplicated infections, 74% showed complete bacteriological cure after a single course of terramycin. An additional 10% showed clinical improvement, although bacteria still grew. Patients with mixed or complicated infections generally failed to respond to a single course of terramycin, but showed clinical improvement with combined streptomycin-terramycin therapy. The majority of Gram-positive organisms tested by the disk method responded to penicillin, aureomycin or terramycin, while most of the Gram-negative organisms were sensitive to streptomycin, chloramphenicol or terramycin. Therefore the range of antibacterial action of terramycin is very wide.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

LXXV.

WHOOPING-COUGH.

THE spotlight in in recent years has fallen so much on diseases like poliomyelitis and rubella that it is easy to overlook the fact that in early childhood whooping-cough or pertussis remains the most serious and fatal of the infectious diseases, with the exception of gastro-enteritis. In spite of a steady decline over the past twenty-five years in both the incidence and the mortality of pertussis, deaths from this disease in the first two years of life still exceed the combined total of those from scarlet fever, measles and diphtheria. At Fairfield Infectious Diseases Hospital in the past decade pertussis has caused six times as many admissions of children under two years of age and has shown a mortality rate six times as high as has the dreaded disease misnamed infantile paralysis. Despite the introduction of immunization procedures, admissions for pertussis in 1951-1952 exceeded those for any other year since 1940-1941. For these and other references to recent experiences at Fairfield Infectious Diseases Hospital, the writer is indebted to the medical superintendent, Dr. H. McLorinan, and the consultant physician, Dr. S. W. Williams. In this article attention will be focused on those aspects of epidemiology, diagnosis, treatment and prevention in which recent work has added knowledge of particular importance to the clinician.

Epidemiology.

Formerly a disease of winter epidemics, pertussis has now become an endemic disease like scarlet fever, occurring in all seasons. In recent years its peak month has been in spring or summer as often as winter.

It occurs at all ages, with its greatest incidence in toddlers and in six-year-olds. As Bordet noted in 1906, it is not prevalent in the first months of life; but it has been diagnosed a number of times as early as the first fortnight, with a very high mortality. The susceptibility of newborn infants is of increasing importance. Fewer mothers, probably less than 15%, now reach term with a significant level of circulating antibodies in the cord blood; these are seldom enough to provide full immunity, and they disappear within a few months. In older children and adults pertussis may pass unrecognized because the characteristic whoop is absent. Grandparents in an infected household are particularly apt to contract it, sometimes in a severe form. Second attacks do occur. Immunity is not necessarily permanent, and it may be quite low after an attack treated in the early stages with antibiotics. A further problem has arisen with the discovery that carrier states exist, particularly in contacts who have been previously immunized.

The American Public Health Association regards the period of infectivity as from seven days after exposure to three weeks after the onset of typical paroxysms. In as many as 12% of cases, however, positive results may be obtained from swab cultures in the sixth week of the illness. In Australia the regulations now in force in each State require that a patient be excluded from school until provided with a medical certificate of non-infectivity, or, if a medical certificate is unobtainable, until four weeks from the commencement of the whoop. Contacts are not excluded from school, but are excluded from a kindergarten for twenty-one days if they have not previously suffered from pertussis.

Pathology.

Hæmophilus pertussis is the organism usually responsible for this disease. Reports from overseas indicate that a closely related bacillus, *H. parapertussis*, is responsible for a small proportion of mild cases of pertussis, but there is only one report of its identification in Australia (Fisher). There are also rare cases caused by *Bacillus bronchisepticus*. The existence of these last two organisms is of some clinical importance because infection due to them does not immunize the patient against *H. pertussis* infection.

In mild cases there is a superficial inflammation of the trachea, bronchi and bronchioles, with some peribronchitis. *H. pertussis* organisms are present on and between the cilia. In more severe cases the bronchioles are predominantly affected, with peribronchiolitis; in bronchopneumonic areas the organisms are also found in the alveoli.

The characteristic toughness of the bronchial mucus often seems to be responsible for the blocking of some of the bronchioles, leading to what is a frequent finding in the paroxysmal stage—collapse of one or more segments, lobules or lobes of the lung. Collapsed areas are usually in the lower lobes, or in the lingula or right middle lobe. Bronchopneumonia is preceded by peribronchiolitis or by secondary infection in collapsed areas.

Minute scattered hæmorrhages in the brain are thought to be common, occasionally causing transient pareses, but gross cerebral hæmorrhage is comparatively rare. In patients who have died with convulsions examination of the brain shows congestion and œdema, sometimes with cellular infiltration.

Clinical Picture.

A full description of the clinical picture would be out of place in this article. By the end of the second week in the average case the cough is predominantly nocturnal and becoming spasmodic, and in the third week troublesome paroxysms occur with vomiting, loss of sleep and usually loss of weight. By this stage the characteristic whoop is evident in young children, though it is seldom heard in later childhood and may be absent in infants under three months of age. Anorexia and loss of weight may be severe in infants and frail children. Coughing often causes ulceration of the *frenum linguæ*.

Recovery usually begins three to four weeks after the onset of the cough, and is complete within another month. In some children, however, the paroxysms may persist for a long time, and they may recur after chilling and during measles and upper respiratory tract infections. *H. pertussis* cannot be cultured during these paroxysms, and so this has been referred to by some as the "neurotic or psychic stage", the typical cough being attributed to a pathological conditioned reflex established during the original infection.

Mild and even abortive forms occur, especially in adults and after immunization. The cough is sometimes partly replaced by paroxysmal sneezing.

Complications and Sequelæ.

Complications and sequelæ occur far more frequently in younger children. Serious complications are found almost entirely in allergic patients and in those debilitated by prematurity, congenital heart disease, malnutrition, recent illnesses or old age.

By far the most frequent complication is bronchopneumonia. Banks holds the view, not universally accepted, that this is nearly always associated with pulmonary collapse. With any patient who in the paroxysmal stage develops listlessness and anorexia, a tinge of cyanosis in the lips, increased respiratory rate or occasional elevation of temperature, areas of pulmonary collapse and/or pneumonia should be suspected, and both postero-anterior and lateral X-ray pictures of the chest will be desirable. It is important to remember that with the onset of pneumonia the paroxysms may diminish, and that in infants there may be no pyrexia. Emphysema, sometimes extending into the neck and axilla, is an infrequent but spectacular occurrence. Right-sided cardiac failure is an occasional complication. Of the sequelæ, bronchiectasis causes the most invalidism. It usually follows unresolved pneumonia or persistent pulmonary collapse, and is most extensive in frail or allergic young children, and in those in whom pertussis and measles have been coexistent.

Convulsions are important as one of the most common immediate causes of death. Though mononuclear infiltration of the brain and meninges may be found in some fatal cases, severe convulsions are usually associated with extensive bronchopneumonia and pulmonary collapse, and some authorities consider that the convulsions are due mainly to anoxæmia. Intracranial hæmorrhage is rarely serious, as has been discussed above. The mechanical strain of coughing may cause subconjunctival and nasal hæmorrhages, and in infants rectal prolapse and hernia.

Nervous sequelæ, such as ataxia, a low threshold for convulsions, mental retardation and personality changes, are reported by some workers, but their relationship to pertussis has yet to be proved.

Enteritis, encephalitis, stomatitis *et cetera* are included in most large series as complications of pertussis, but their incidence suggests that they may be more correctly regarded as intercurrent infections.

Diagnosis.

The diagnosis can be suspected if the cough is of gradual onset, is worse at night and is becoming paroxysmal, and especially if the eyes water and the face goes red in a paroxysm and if this ends with a typical whoop and is followed by vomiting, or if the frenum of the tongue is ulcerated. There are, however, many atypical cases, especially in immunized subjects, and 100% results cannot be expected from laboratory tests.

The best cultural results are obtained with the pernasal naso-pharyngeal swab, which has superseded the cough plate and the peroral swab. The child lies on his back, the head held firmly by an assistant. A fine dry sterilized swab stick is passed along the floor of the nose and pushed back until it touches the posterior pharyngeal wall. To ensure that the swab will not be left behind in the nose, it is preferable to use a wire stick to which the wool has been sealed with collodion. Culture must be made on Bordet-Gengou or other medium within twenty-four hours, and preferably within six to twelve hours. To get the best results the swab should be taken during or just after a paroxysm. If necessary, a paroxysm may be induced by placing the thumb in the child's suprasternal notch and with the other hand extending the head fully and then flexing it sharply so that the thumb rides over the child's trachea. By this method, positive cultures are obtainable in 80% of cases in the first week, and in at least 60% in the paroxysmal stage.

In the earlier weeks investigation with the naso-pharyngeal swab is far more often diagnostic than the lymphocyte count. The normal infant in the first year has as many as 6000 to 12,000 lymphocytes per cubic millimetre. A pathological lymphocytosis is usually not present before the paroxysmal stage, but it may be helpful in diagnosis then if the swab findings are negative.

Infection with *H. parapertussis* or *B. bronchisepticus* is not recognizable without advanced laboratory techniques, which as a rule are not warranted. It is, however, necessary to differentiate pertussis from other acute conditions that may produce a spasmodic cough—influenza, measles, laryngotracheitis, bronchitis—and from more chronic conditions, such as bronchiectasis, aspirated foreign body, tracheobronchial lymphadenitis and tuberculosis.

Prognosis.

A steady fall in mortality from pertussis has occurred over the past quarter of a century, running parallel to the decline in mortality from pneumonia. It is hard to estimate the mortality rate from pertussis owing to incomplete reporting of cases, but it is probably less than 0·5%. Cases in which the patients were treated in hospital today show an overall mortality rate of 2% to 3% because they include a high proportion of cases with complications. For some reason pertussis, unlike other acute infectious diseases of childhood, has a lower mortality rate in boys than in girls, and this low masculinity is reflected in bronchiectasis (Lancaster, Dods).

Although pertussis has its highest incidence in the second and later years, its morbidity and mortality are highest by far in the first year and higher still in the first six months. At Fairfield Infectious Diseases Hospital in the past decade 86% of the deaths occurred in the first two years of life; there were 48 deaths in the first year compared with 19 in the second, the respective mortality rates being 14·5% and 8·6%. Even with modern improvements in treatment and prophylaxis, the mortality rate in children in hospital under twelve months of age is at least 5%.

Experience shows that, as a frequent cause of bronchiectasis, pertussis is second only to pneumonia. The incidence of this sequela is related to the age and condition of the patient when he contracts pertussis, to the severity of the attack, and particularly to the care taken in the early diagnosis and thorough treatment of pulmonary collapse and bronchopneumonia.

Treatment.

General Measures.

Fresh air and sunshine are beneficial, and both rest and exercise have their place in treatment. If the appetite is poor, if the temperature is raised, or if cough and vomiting are severe, bed rest is advisable, but the average apyrexial patient should not be kept in bed. Frequent small meals of readily digestible food help to prevent wasting in a vomiting child. For infants, breast milk is preferable; the mother of an infected newborn should accompany her infant if he is admitted to hospital. Gavage feeding is contraindicated.

The temperature should be taken twice daily if there are any doubts about the child's progress, especially in the paroxysmal stage, when pulmonary complications are likely to develop. It is important, in infants particularly, to prevent contact with persons suffering from respiratory tract infections or measles.

Symptomatic Treatment.

Drugs which reduce the force of the paroxysmal cough have the disadvantage of increasing the tendency to pulmonary complications through retention of mucus. However, provided the patient is not cyanosed, phenobarbitone, one-quarter to three-quarters of a grain, at 6 p.m., is worth prescribing to encourage sleep and thus prevent loss of weight. Expectorants should not be given as a routine because they often increase vomiting, though they can be helpful in the later weeks. In infants atropine methylnitrate ("Eumydrin") may be useful in lessening vomiting, given as a 0·6% alcoholic solution in a dosage of two to six minims four-hourly before meals. Allergic children with bronchial spasm may be helped with one-quarter to one-half grain of ephedrine three times a day combined with four to eight minims of tincture of stramonium. Antihistamine drugs have also been reported to be beneficial (Verrotti), and for a very sick allergic child the merits of a short course of ACTH or cortisone are worthy of consideration. When bronchial mucus is abundant and the expulsive powers are weak, as in small infants, aspiration and postural drainage are indicated.

Specific Treatment.

Rapid immunization of contacts in the incubation period with three doses of pertussis vaccine at four-day intervals has been advocated by Sauer, but there is no convincing evidence of its value except perhaps in boosting the immunity of children previously immunized or of adults who may possess some degree of basal immunity. There is little doubt that antibiotics will prove more helpful than vaccines in this early stage.

No appreciable benefit is produced by sulphonamides, penicillin or streptomycin, except in the control of secondary infections. A more specific effect, however, is obtainable with the most recently discovered antibiotics—aureomycin, chloramphenicol, terramycin and others not yet available in Australia. They can be relied upon to produce negative findings from naso-pharyngeal swab investigations in from a few days to two weeks, and, of course, they will reduce the incidence of secondary infections. In addition it is an almost universal experience that they improve the well-being of the patient and greatly reduce the severity and the frequency of the paroxysmal cough within a week, reduce temperature and vomiting, and shorten the duration of the illness. Given in the incubation period or in the pre-paroxysmal stage, they will abort or arrest the great majority of cases. There is little to choose between the three antibiotics mentioned, though aureomycin appears slightly less effective than terramycin and chloramphenicol. Considering the rare development of aplastic anæmia following the use of chloramphenicol, the drug of choice at the moment seems to be terramycin. The appropriate dosage is practically the same for all three antibiotics. Although some workers advise as much as 100 to 120 milligrammes per kilogram of body weight *per diem*, the minimum effective daily dosage is about 50 to 60 milligrammes per kilogram of body weight, or 25 milligrammes per pound. For an infant aged twelve months weighing about 20 pounds, this amounts to a daily dosage of 500 milligrammes. This can be given in three or four equal divided doses, but there are reasons for making the bedtime dose the largest one. A great boon to clinicians, parents and young patients has been the introduction of new palatable preparations: "Chloromycetin Palmitate" (Parke, Davis and Company) containing 125 milligrammes per drachm, and "Oral Suspension of Terramycin" (Pfizer) containing 1500 milligrammes per ounce. Unfortunately these preparations are not inexpensive, and they have not as yet been listed under the *Pharmaceutical Benefits Act*. The course of treatment usually recommended is for about ten days, but relapse somtimes occurs if the course ends before the fourth week of the illness.

Should these antibiotics be given in every case? Most emphatically the answer is "no". Apart from their cost and the difficulties of their administration, they have other important disadvantages. They all produce changes in the bacterial flora of the naso-pharynx which may lead to pneumonia caused by a resistant organism, and they may produce similar changes in the alimentary tract resulting in vomiting, diarrhœa and excoriated buttocks. Most arresting is the fact that after an attack treated with antibiotics the blood of the patient has such a low titre of protective antibodies that he may be susceptible to reinfection and so need a course of pertussis vaccine. Furthermore in some hospitals doubt has been cast on the value of these

"specific" drugs. There appear to be two reasons for this: first, their effectiveness is maximal in the early stages, whereas in many cases the patients are admitted to hospitals only in the later stages, when a dramatic result cannot be expected; secondly, when unfavourable results have been reported, the dosage of the antibiotic has usually been below the effective minimum.

To summarize: the antibiotics should be prescribed only in cases in which the prognosis is likely to be poor—in severe attacks, in the presence of complications, for debilitated patients and for infants. In the warning words of Freeman, "these antibiotics are a magnificent life-line to be thrown to those struggling in deep waters; but, if our patient is liable to tumble in again, he had better perhaps be taught to swim out again by immunization".

Treatment of Complications.

Bronchopneumonia is usually best managed in hospital, the treatment including oxygen and antibiotics as for bronchopneumonia from other causes. The patient with pulmonary collapse, if free from pyrexia, listlessness and anorexia, should be up and about. However, if signs of secondary infection appear, he will need careful treatment including antibiotics and postural drainage. Any patient with collapse persisting beyond two to three weeks should be referred to a specialist in the hope of preventing bronchiectasis. Convulsions are treated with oxygen and warmth and, if persistent, with lumbar puncture, with sodium phenobarbitone by intramuscular injection (half a grain for an infant of one year) or paraldehyde *per rectum*, or with "Phenytoin" or "Pentothal".

Prevention.

Isolation.

The aim should be to postpone pertussis until after the fourth year, or later in frail or allergic children. Whenever practicable, in suspicious cases the affected person should be isolated early from infants, young children and elderly people. It is never justifiable to expose a newborn infant to infection, a fact which needs to be considered by the organizers of ordinary hospital and health centre clinics. The regulations concerning isolation and quarantine are referred to under "Epidemiology". In highly susceptible contacts, such as unimmunized infants, the disease may be aborted or mitigated with antibiotics given in the incubation period, or less effectively with rapid immunization or with pooled human serum (20 mils).

Immunization.

Successful active immunization by vaccines was introduced by Sauer in 1933. Useful later innovations include the use of a mineral adsorbent (Harrison, 1938), immunization in early infancy (Peterson, 1942), ante-natal immunization (Cohen, 1943), a fluid-grown vaccine (Wilson, 1945), and an adsorbed hæmagglutinin vaccine (Keogh, North and Warburton, 1947). The controversy which raged, especially in British countries, about the value of pertussis vaccines has now been settled by a well-controlled large-scale field trial conducted in England for the Medical Research Council. Over an observation period of two to three years, the incidence of pertussis in unimmunized children was from three to ten times as high as in immunized children, and when pertussis did occur in the latter group it was on the average of less severity and of shorter duration. With the best adsorbed and fluid-grown vaccines, such as are now being produced by the Commonwealth Serum Laboratories, it is likely that even better results may be obtained.

Concerning the optimum time for immunization and the most suitable type of vaccine, opinions still differ. It is universally agreed, however, that early immunization is essential to prevent the high death rate in the first year of life. Immunization of the mother during pregnancy will give the infant an appreciable though rapidly waning immunity for the first few months of life, but there are too many shortcomings and disadvantages in this procedure to make it advisable as a routine. Immunization of the infant commencing at one month of age is considered effective provided an adsorbed vaccine is used, such as hæmagglutinin, aluminium phosphate adsorbed (H.A.P.A.), produced by the Commonwealth Serum Laboratories. Although pertussis is infrequent below the age of three months, this method is valuable where very early immunization seems desirable. Unfortunately, this age is too early for effective simultaneous immunization against diphtheria. For general use, therefore, the growing opinion of authorities in Australia favours a pertussis vaccine combined with diphtheria toxoid, given in three injections at monthly intervals, commencing at three or four months of age. The diphtheria antibody response at this age may not be as great as with immunization carried out after the sixth month, so a small booster injection of diphtheria toxoid is usually advised at the age of twelve to fifteen months. This minor disadvantage is well compensated for by the fact that immunization conducted in the first six months carries with it virtually no risk of paralysis from concurrent poliomyelitis. For some years suitable proprietary combined vaccines have been imported and extensively used in Australia. Recently a similar preparation called "Mixed Pertussis and Diphtheria Antigen" was produced by the Commonwealth Serum Laboratories, and before long this same vaccine with the addition of tetanus toxoid ("Triple Antigen") will also be available. The fact that the pertussis component of these Commonwealth Serum Laboratories mixed vaccines is prepared in a simplified liquid medium instead of on the usual solid Bordet-Gengou medium is considered likely to enhance its potency. For the dosage and other details concerning these various vaccines, the reader is referred to the pamphlets provided with them and to two articles by North appearing during the year in this journal.

Reactions have always been a greater hazard with pertussis vaccines than with most other vaccines, but their incidence has been greatly reduced with these latest preparations, and they are usually extremely mild, provided the precautions advised are taken. A family history of fits or convulsions, of mental defect or of allergic disorders is always an indication for caution, and in these cases any reaction may be taken as a contraindication to further injections. Immunization should be deferred in any child who is not in good health, even if he is afebrile and only seems "off colour" or "off his food" and not gaining weight well. Injections must be given subcutaneously and into a different site each time, preferably in the upper arms. It is essential that the infant be held firmly. The mother should be informed of the possible reactions, and she should be told to reduce the size of the next meal if the child seems hot, to offer plenty of fluids, and to give half a tablet (two or three grains) of aspirin if he is fretful. Any reaction that is not already subsiding within thirty-six hours should be reported to the doctor.

JOHN H. COLEBATCH,
Melbourne.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

A NEW INSTRUMENT.[1]

[*The Australian Medical Gazette*, May, 1871.]

ONE of the Melbourne papers (Argus) in an obituary notice of the late Mr. Hugh Glass contained the following remarkable passage in its issue of 16th inst.: "His (Mr. Glass's) ordinary medical attendant, Dr. Martin of Collins St., having visited Europe, brought back with him, a few months since, a new instrument for opening cystic tumours of the liver and this was applied in Mr. Glass's case, the result being, we are informed, to show that the disease was of a form not to be *effectually* relieved by such means." From the wording of this paragraph, it may be fairly inferred that the information so considerately conveyed to the public in the foregoing passage was supplied to the press by the fortunate importer of the "new instrument". In return for imparting so much confidence and valuable information, the grateful journalist could not do less than give his informant the benefit of a gratuitous advertisement by way of a *quid pro quo*. It is rather unfortunate that, at the after-death examination instead of being affected with cystic disease, "the liver and right kidney of the deceased were found to be enormously enlarged and studded with cancerous disease almost throughout". Tapping a cancerous liver with an instrument for opening cystic tumours of the viscus, if unattended with any other advantage, possesses, at all events, the negative merit of being novel and sensational. It is not much calculated to enhance the value of the "new instrument" or to extend

[1] From the originals in the Mitchell Library, Sydney.

the fame of its enterprising importer to find that its *début* in this colony was coincident with a blunder. Seriously, every right thinking medical man must deeply regret that the highly unprofessional and reprehensible practice of procuring the insertion of newspaper paragraphs detailing the treatment of cases of disease, vaunting the success of operations, or eulogizing the wonderful skill of some member of the profession, prevails too much in this colony and especially in the metropolis. No professional man, possessed of great merit or actuated by right feeling, would resort to such unseemly and derogatory manœuvres. It seems strange that the *Australian Medical Journal*, which is so great a stickler for obsolete and useless distinctions in the profession, has not a word to say against a system of arrant puffery which should be scouted by every reputable member of the profession.

Obituary.

CHARLES VINCENT MacKAY.

We are indebted to Sir Victor Hurley for the following appreciation of the late Dr. Charles Vincent MacKay.

The recent death of Dr. Charles V. MacKay will be especially deplored by those medical graduates who passed through the medical school of the University of Melbourne in the few years prior to the first World War. He was born in 1880 and, after gaining a Government scholarship, attended Hawthorn College for three years. After leaving school he worked for a short period as a bank officer, and also on the land. He then commenced his medical course and graduated at the final examinations in 1905. He gained first-class honours in medicine and in surgery with a high place on the final honours list, qualifying for appointment in 1906 as a resident medical officer at the Melbourne Hospital. In the following year he was a resident medical officer at the Children's Hospital, and then, after a short period as medical superintendent of the Infectious Diseases Hospital, he was appointed medical superintendent of the Melbourne Hospital in 1908, in succession to the late Sir John McKelvey. He occupied this position until 1912, and during this period Sir Hugh Devine, Dr. M. D. Silberberg, the late Sir Alan Newton and the writer were registrars under him at the hospital. His four years as medical superintendent were very onerous ones, as it was during this period that the hospital was rebuilt on the Lonsdale Street site, to be transferred thirty years later to its present site at Parkville. He obtained his M.D. degree in 1910. Between 1912 and 1914 he was a clinical assistant and later was honorary physician to outpatients at the hospital, and he practised as a physician in the city.

At the outbreak of the first World War he went to England and joined the Royal Army Medical Corps. He was placed in charge of the medical division of the newly established King George Military Hospital in London. Later he was promoted to the rank of lieutenant-colonel and served on the Salonika front, where he was in command of the Number 80 British General Hospital. After the end of the war he remained for some years in England. He had always taken a special interest in orthopædics and in the disabilities resulting from infantile paralysis. In this he was closely associated, both in England and in Australia, with the work of his cousin, the late Sir Colin MacKenzie. He was appointed to the position of officer in charge of the muscle reeducation department, Alder Hey Hospital, Liverpool, which was the forerunner of the more highly organized rehabilitation centres of the second World War. Later he was for some years an orthopædic specialist and Deputy Commissioner of Medical Services to the Ministry of Pensions, London. For the next fourteen years he practised in Wimpole Street.

He returned to Australia in 1936, when he was appointed medical assistant to Sir Colin MacKenzie, who was then Director of the Australian Institute of Anatomy at Canberra. From 1939 to 1942 he occupied the position of executive medical officer to the Anti-Cancer Council of Victoria. From 1942 to 1945, during the second World War, he became executive medical officer of the Medical Equipment Control Committee, then under the direction of the late Sir Alan Newton. After the end of the war until his death he was associated with the Cancer Institute, first as secretary, and later as executive medical officer. He published various papers on orthopædic subjects and on muscle reeducation, in which he was associated with the pioneer work done in this field by Sir Colin MacKenzie. He was editor of the latter's book "The Action of Muscles".

Beneath a shy and reticent exterior Charles MacKay possessed qualities of sound judgement and a firm resolution in maintaining principles which he thought were right. He had a stammer which was a great handicap to him during his medical course and for some time afterwards, but by sheer determination he overcame it. Slow and deliberate in speech and action, he was a sound and able administrator in his various activities. As a comparatively young man in charge

of a large hospital in an important stage of its development, he gained the confidence and appreciation of the medical staff as well as of the members of the committee of management by the competent way in which he dealt with knotty and difficult problems as they arose. He was rather aloof from the younger members of the resident medical staff, but took a great personal interest in the careers of all those who passed through his hands, and gained greater pleasure in their successes than in anything he accomplished himself. Invariably kind and considerate in his dealings with patients, loyal and dependable in his many friendships both within and outside the profession, Charles MacKay will be held in affectionate remembrance by the large circle of people with whom he came in contact. His brother, Dr. Eric MacKay, is one of the senior medical officers in the Repatriation Department.

Correspondence.

THE USE OF ANTIBIOTICS.

Sir: It is now certain that antibiotic-resistant strains of organisms do occur. This antibiotic resistance is seen chiefly in hospitals and is closely linked with the problem of cross-infection. Many organisms, especially strains of *Staphylococcus pyogenes* and strains of the Gram-negative bacilli, have developed resistance against all the antibiotics in common use—namely, penicillin, streptomycin, aureomycin, terramycin and chloramphenicol.

Two new antibiotics have recently been made available under the names erythromycin ("Ilotycin", "Erythrosin") and carbomycin ("Magnamycin"). These antibiotics have a spectrum of activity similar to that of penicillin in that they are effective against Gram-positive bacteria. One of them, erythromycin, is also effective against *Hæmophilus pertussis*. It has been shown that organisms will develop

resistance against these two antibiotics although no cross-resistance has been demonstrated with penicillin, streptomycin, aureomycin, chloramphenicol and terramycin as yet.

In these two new antibiotics we have a weapon to treat those cases of infection due to organisms resistant to all other antibiotics; cases which at present are often fatal. If the use of these new antibiotics is restricted to the treatment of such cases their value will be enhanced, their period of usefulness prolonged and the appearance of resistant strains delayed. If they are used without thought very soon resistant strains will develop and once again there will be cases of infection for which nothing can be done.

Is it too much to ask the medical practitioner to use erythromycin and carbomycin only when absolutely necessary and if possible only after adequate laboratory investigation of the case?

As strains of organisms resistant to the antibiotics already in general use are most common in hospitals, it would seem that the main sphere of usefulness of these new antibiotics will be in hospitals where adequate control can be achieved.

Yours, etc.,
EDGAR THOMSON, Director.

Fairfax Institute of Pathology,
Royal Prince Alfred Hospital,
Camperdown, New South Wales.
August 4, 1953.

TEACHING METHODS.

SIR: Not so long ago in these pages Dr. Kempson Maddox drew attention to the need of elementary instruction in the basic principles of methods of teaching for those members of the medical profession who have the important task of instructing medical students. This need applies both to those who lecture in our university classrooms and to those who teach in our various hospitals. An example is afforded in the otherwise admirable contribution published in the issue of August 1 by Dr. Claffey and Dr. Newton, "Acute Infections of the Fingers and Hand". After pointing out that some teachings which appear in most standard text-books are erroneous, the authors include two diagrams illustrating methods which they describe as wrong and dangerous. A false visual impression is liable to persist and is a cardinal error in teaching.

Yours, etc.,
A. W. J. BULTEAU.

3 Plunkett Street,
Kirribilli,
New South Wales.
August 4, 1953.

COMPULSORY CHEST X-RAY EXAMINATIONS.

SIR: Amidst much that is of sound value in the thoughtful letter of Dr. B. Short of June 26, 1953 (M. J. AUSTRALIA, July 11, 1953), are certain views of which consideration gives rise to a feeling of uneasiness.

He states, for example: "After all, tuberculosis has some symptoms and makes people sick. If it does not cause a cough or ill-health in some form, it requires no treatment." Coming from a medical man, in times when many members even of the laity are aware that the disease can be present in an active form without giving rise to appreciable symptoms, this is, to say the least, a somewhat unexpected statement. This surely is the stage at which to assess the need for that early treatment for which Dr. Short so ably pleads elsewhere in his letter. Careful observation, and possibly a modification of their environment, is the least duty we owe to such patients.

Furthermore, one is all too familiar with that recurring situation in which the disease has reached a surprisingly advanced stage before obtruding itself upon the unfortunate patient's notice. Varying individual "thresholds" to symptoms form a commonplace of medicine, and asymptomatic disease of almost any kind is not necessarily synonymous with early disease.

And if we are to wait until symptoms develop, what then? How often do people misinterpret and therefore disregard the early symptoms of tuberculosis? Tiredness or weight loss are ascribed to overwork or worry, cough to excessive smoking, and so on.

I do not know of any physician who nowadays relies upon a single X-ray film *per se* to diagnose active tuberculosis. In any case, such a shortcoming could never serve as a valid criticism of the method of mass radiography, the function of which is surely to discover an abnormality. Information as to its nature and status is gained by subsequent investigation. Incidentally, tuberculosis is, of course, not the only disorder which may thus reveal itself; symptomless bronchogenic carcinoma is an example of another.

Most would agree with Dr. Short's advocacy of selective mass radiography, providing the general public is not excluded from ultimate participation in the scheme, but it may be doubted whether the increased organization which this modification would demand could be achieved with the resources at present mobilized.

I believe that most of us who have to deal with this problem are actively aware of the danger of generating morbid anxiety. This may be done by over-emphasizing those unimportant or minor deviations from the normal sometimes revealed by chest skiagrams. It is axiomatic that on such occasions a thorough investigation should be followed by full explanation and reassurance, emphatically given.

On the other hand, over-emphasis of this danger, or any other consideration, should not lead us to adopt the ostrich with buried head as the device on our battle-standard. We are not looking for trouble, but we are engaged with a relentless and unpredictable foe.

Yours, etc.,
R. H. BRENT.

27 Glendower Avenue,
Eastwood,
New South Wales.
July 17, 1953 (received August 10, 1953).

SIR: From their replies to Dr. Short's letter in the journal of August 1, it is obvious that Dr. Rubinstein and Dr. Wilson do not realize that Dr. Short, in his whimsical way, is endeavouring to draw attention to the deficiencies in the present set-up for the diagnosis and treatment of tuberculosis, particularly as it exists in New South Wales today. He is not attacking mass X-ray surveys alone.

In the United States of America it is now being advocated that instead of mass X rays, all patients should be X-rayed as routine on admission to hospital irrespective of the condition for which they are being admitted. The National Tuberculosis Association of America states that figures show that by doing this the case-finding percentage is approximately double that obtained from mass X rays. Such a method would be a far better and certainly a far more economical alternative to such surveys as that carried out in the country districts by the Anti-Tuberculosis Association of New South Wales between 1950 and 1952, when only 59 active cases were found out of 138,732 examinations.

Everyone will agree that to eradicate tuberculosis from this country all methods of investigation must play their part, and to ensure this, there must be complete cooperation between all bodies engaged in this work. It is equally important to have Mantoux surveys, as are at present being carried out by the New South Wales Health Department, as it is to have mass X-ray surveys. Also NAPTA regards education of the public in regard to the disease as a vital aid. Epidemiological and sociological research and rehabilitation are other necessary avenues to be completely developed.

Whilst not agreeing with most of Dr. Short's statements, yet it is felt by my Association that if this correspondence has only focused the eyes of the medical profession on the many aspects of the problem of tuberculosis control, it will have more than served its purpose.

Yours, etc.,
DAVID A. HUGHES,
Honorary Secretary, National Association for the Prevention of Tuberculosis in Australia (New South Wales Division).

Sydney,
August 5, 1953.

CARDIAC ARREST AS A SURGICAL EMERGENCY.

SIR: Cases of cardiac arrest during operations may be subdivided into two distinct categories: (*a*) Those occurring during an operation in which the abdomen or chest is open and the heart beat appears to have stopped and massage through the diaphragm or pericardium starts it contracting again, and after perhaps a period of anxiety, the circulation remains satisfactory and the operation is completed and the patient recovers none the worse for the incident, and (*b*) when the foregoing fails, or, as in the case of an orthopædic or ear, nose and throat operation, neither the abdomen nor chest is open, or else one is called to a theatre for assistance and confronted for the first time with a patient whose heart has stopped.

In the second type of case prompt action is imperative and it is necessary to have a definite plan.

The following is suggested as an alternative to the procedure outlined by Ian Monk (M. J. AUSTRALIA, August 1, 1953, page 170):

1. The anæsthetist institutes artificial respiration.
2. The patient's head is lowered to minimize cerebral anoxia, but the body is kept horizontal.
3. If not already "washed up" it takes a few seconds to put on sterile gown and gloves while a nurse swabs the epigastrium with antiseptics. A mid-line incision is made, and the diaphragm is incised. This approach is equally or even more rapid than incising the chest wall and has not the disadvantages of opening the pleura. It gives ready access to the heart and is not an unduly uncomfortable position for the hand to maintain massage.
4. The ventricles are grasped, and massage is commenced by alternately squeezing and relaxing. In favourable cases vernicular or fibrillary contraction is eventually replaced by systole and diastole, which is at first feeble but may improve when oxygenated blood reaches the myocardium. When cardiac anoxia is overcome and any toxic substances are washed out, the heart beat may continue without assistance.
5. The legs are then suddenly raised to the vertical position. This aims at raising the pressure in the *vena cava* and providing the necessary stimulus to more forcible contration, and this manœuvre may produce a considerable increase in force.
6. The question of using drugs or a transfusion may then be considered.
7. If all goes well the wound is sutured. It is not essential to close the aperture in the diaphragm, as it is effectively blocked by the left lobe of the liver.
8. The patient is returned to the ward and subsequently treated according to progress.

Though the heart has been restored by extradiaphragmatic massage, I would agree with Dr. Monk in his valuable paper that it is an inefficient method, and no time should be wasted in persisting with it.

Few surgeons who work with competent anæsthetists are likely to have any very extensive personal experience with cardiac arrest during operations, and the subject is therefore admittedly likely to remain controversial. The futility of attempting to restart the heart by injecting drugs needs emphasis because I believe this is still often done, and in any case the value of drugs seems to be limited. I would regard the act of raising the legs to give the heart more blood to contract upon to be of considerable value, and the opportunity of doing this is lost if the table is put into the head-down position at the outset with the object of limiting cerebral anoxia.

Massage must restart effective circulation within a minute or two, otherwise apparent success is likely to be followed by the chagrin of observing the patient remaining in a decerebrate condition and succumbing a few hours or days later.

Can anybody devise a more appropriate term than cardiac "massage"?

Yours, etc.,

F. STONHAM.

2 Collins Street,
Melbourne, C.1,
August 8, 1953.

ACUTE INFECTIONS OF THE FINGERS AND HAND.

SIR: Dr. Claffey and Dr. Newton are to be congratulated, not only upon the lucidity of their article concerning acute infections of the hand and fingers which appeared in THE MEDICAL JOURNAL OF AUSTRALIA of August 1, but also upon their excellent results achieved in their clinic at Saint Vincent's Hospital.

It is to be regretted, however, that they did not refer to the original teachings of Böhler, of Vienna, whose article upon the treatment of hand infections appeared (I have forgotten exactly when and in what journal) some time prior to 1937. He, in his turn, apparently adapted his technique from the teachings of Winnet-Orr and Trueta. His teachings were (i) adequate anæsthesia, (ii) the application of a tourniquet, the reason of which was to permit the surgeon to observe exactly the extent of the suppuration, (iii) an incision extending well beyond the suppurative process, (iv) packing the incision with a wick of "Vaseline" gauze, and (v) immobilization of the affected part. The dressing was left untouched for a week, progress being noted by inquiring about throbbing, rise in temperature and noting if any swelling occurs proximal to the affected part.

My partners at that time adopted those teachings, and, to the best of my knowledge, with no untoward results with the exception of definite apprehension on the part of nursing staffs. The introduction of antibiotics has only resulted in one major change in treatment: namely, that in early acute infections expectant treatment could be safely adopted; this particularly applied to infections of the tendon sheaths.

Might I be excused for amending their description of erysipeloid infections? Practitioners in my area, until recently, came into contact with many of these cases. In addition to other types of infections of the fingers and the foot it was a very common disease of the wool-washing industry. Popularly called skin poisoning, men employed at "pie" picking (piece picking) were particularly prone to it. A peculiar fact, not mentioned in text-books and brought to my notice by these men, was that one attack conferred immunity for many years. Men suffering from this infection first complain of pain in the affected part (hand or foot); and when it first appears in an end phalanx a differential diagnosis from a pulp infection is extremely difficult. Most practitioners have opened at least one finger. In normal cases the colour of the affected part is bright red (hence its name). In more severe cases the colour darkens, whilst in very severe cases it becomes almost purple; in these cases suppuration, quite superficial and only in the form of blisters, occurs, the pus being dirty in colour and quite foul. This infection has a peculiar tendency to travel, often from finger to finger; it may at times "jump" one finger. After trying all types of treatment I finally adopted "Tincture Metaphen"; but I am not at all sure that this may act only as a placebo, the infection of its own accord dying out.

Yours, etc.,

C. H. JAEDE.

Mascot,
New South Wales,
August 2, 1953.

SIR: I am grateful to Dr. Claffey and Mr. Newton for their article on "Acute Infections of the Fingers and Hand". Our practice involves seeing many such infections, for the most part in injured industrial workers, and this article confirms one's ideas on some aspects of this subject and gives fresh ideas on others.

There are two or three things which call for comment. Firstly, the statement that "no incision is made unless there is evidence of localized pus formation". Though this is true, I find it hard to tell sometimes when pus has formed. The teaching of a prominent Victorian surgeon that when an infected finger keeps a patient awake it is time for the finger to be opened, is a very useful rule, with few exceptions. Secondly, the statement that "only the base of the nail or its lateral margin is resected, if this is undermined with pus". Frequently this is sufficient, but I submit that two things may happen: (*a*) the infection will reappear under the cut edge of the nail with renewed discharge of pus and formation of granulation tissue, or (*b*) the new nail may grow before the remaining portion of the diseased nail comes off and the normal growth of the new nail is interfered with. Therefore it is better, in most cases, to remove the whole nail at the first operation. Thirdly, though agreeing that erysipeloid is a relatively common infection, I am not sure that its course is "benign", as it can be quite painful, and it can disappear in some areas and reappear in adjacent areas for weeks unless treatment with large doses of penicillin is undertaken.

Yours, etc.,

B. H. E. BARRACLOUGH.

The Clinic,
28-32 Parsley Street,
Footscray,
Victoria.
August 7, 1953.

Medical Practice.

THE USE OF ANTIBIOTICS.

THE Antibiotics Committee of the National Health and Medical Research Council has drawn up a table indicating the use of antibiotics in various diseases. It is published herewith at the request of the Director-General of Health, Commonwealth Department of Health, Canberra. Under the headings of the several antibiotic preparations the numbers 1, 2 and 3 refer to the preferences in order in which the drugs should be used, in other words which drug would be expected to provide most benefit in a particular illness. The attention of readers is drawn to a letter by **Dr. Edgar Thomson** appearing in the correspondence columns of this issue.

No.	Disease.	Sulphonamides.	Penicillin.	Aureomycin.	Terramycin.	Chloramphenicol.	Streptomycin.	Erythromycin.	Carbomycin.	Remarks.
1	Hæmolytic streptococcus (group A) infections.	—	1	2	2	—	—	3	3	Resistance to antibiotics not a problem. Use penicillin first. Most cases will respond.
2	*Streptococcus viridans* (endocarditis) infections.	—	1	3	3	—	1	2	2	Combined therapy: penicillin in large doses *plus* streptomycin in doses of two grammes per day up to a total of 20 grammes (adult).
3	*Streptococcus fæcalis* (endocarditis) infections.	—	1	2	2	—	1	2	2	Combined therapy as for number 2.
4	Pneumococcal infections ..	—	1	2	2	3	—	2	2	Resistance to antibiotics not a problem. Should respond to penicillin.
5	Meningococcal infections ..	1	1	2	3	3	—	2	2	Combined antibiotic and sulphonamide therapy.
6	Gonococcal infections ..	1	1	2	2	3	—	2	2	
7	Staphylococcal infections:									Resistance to antibiotics is a great problem, especially in hospitals, where sensitivity tests should be carried out. In general practice penicillin will be effective nearly always, but if there is no response in thirty-six hours other antibiotics should be tried. Sensitivity tests should be done if possible. See note (*d*) below in regard to erythromycin and carbomycin.
	(*a*) In hospitals	—	2	1	1	3	2	2	2	
	(*b*) In general practice ..	—	1	2	2	3	3	2	2	
8	Brucellosis	See remarks.	—	2	2	1	2	—	—	Combined antibiotic and sulphonamide therapy gives best results.
9	Pertussis	—	—	2	2	1	—	—	—	
10	Typhoid fever	—	—	2	2	1	—	—	—	
11	Influenzal meningitis	See remarks.	—	2	—	1	1	—	—	Combined antibiotic and sulphonamide therapy with *Hæmophilus influenzæ* antiserum.
12	Urinary tract infections (uncomplicated);	See remarks.								
	(*a*) *Escherichia coli* ..		—	1	1	2	2	—	—	Antibiotic resistance is a problem in this group. The occurrence of resistant strains follows no set pattern. In hospitals sensitivity tests should always be done. In general practice various antibiotics may have to be tried. Combinations may be found useful. Sulphonamides should be tried first. See notes (*e*) and (*f*) below on polymyxin and bacitracin.
	(*b*) *Bacillus aerogenes* ..		—	1	1	2	2	—	—	
	(*c*) *Proteus vulgaris*		—	2	—	1	1	—	—	
	(*d*) *Pseudomonas pyocyaneus*		—	2	1	—	1	—	—	
	(*e*) *Streptococcus fæcalis* ..		2	1	1	1	3	2	2	
13	Tuberculosis	—	—	—	—	—	1	—	—	Streptomycin should never be given alone. The preferred dose is one gramme twice weekly except in acute cases when one or two grammes daily may be given for a short period.
14	Chancroid	—	—	1	—	2	—	—	—	
15	Friedländer bacillus (pneumonia)	—	—	1	1	2	1	—	—	
16	Salmonella infections (gastro-enteritis and bacteriæmia).	—	—	2	2	1	1	—	—	
17	Bacillary dysentery	1	—	2	2	2	2	—	—	
18	Plague	1	—	—	—	—	1	—	—	
19	Subacute bacterial endocarditis:									
	(*a*) *Streptococcus viridans* (see above).	—	1	3	3	—	1	2	2	Combined penicillin and streptomycin.
	(*b*) *Streptococcus fæcalis* ..	—	1	2	2	—	1	2	2	Combined penicillin and streptomycin.
	(*c*) *Hæmophilus influenzæ et cetera.*	See remarks.	—	2	—	1	1	—	—	Combined antibiotic and sulphonamide therapy.
20	Acute bacterial endocarditis:									
	(*a*) Staphylococcal	—	—	—	—	—	—	—	—	As above under number 7.
	(*b*) Other organisms.. ..	—	—	—	—	—	—	—	—	As above for corresponding organism.
21	Trachoma	—	—	1	—	—	—	—	—	
22	Surgical conditions of bowel (pre- and post-operative).	1	—	—	—	1	1	—	—	Combined antibiotic and sulphonamide therapy
23	Pulmonary conditions (pre- and post-operative).	—	1	1	1	2	2	3	3	Depends on organism causing infection.
24	Typhus and other rickettsial diseases.	—	—	2	2	1	—	—	—	
25	Primary atypical pneumonia	—	—	1	1	1	—	—	—	
26	Psittacosis	—	—	1	2	—	—	—	—	
27	*Lymphogranuloma venereum* ..	—	2	1	1	1	3	—	—	
28	*Granuloma inguinale*	—	2	1	1	1	3	—	—	
29	Anthrax	—	1	2	2	—	—	—	—	
30	Syphilis	—	1	2	2	—	—	—	—	
31	Yaws	—	1	2	2	—	—	—	—	
32	Rat-bite fever:									
	(*a*) *Spirillum minus* ..	—	2	1	—	—	—	—	—	
	(*b*) *Streptobacillus moniliformis*	—	—	—	—	—	1	—	—	
33	Tularæmia	—	—	1	1	—	1	—	—	
34	Gas gangrene	—	1	2	2	—	—	—	—	Combined with antiserum.
35	Influenza	—	—	—	—	—	—	—	—	
36	Common cold	—	—	—	—	—	—	—	—	
37	Infectious mononucleosis ..	—	—	—	—	—	—	—	—	
38	Actinomycosis	See remarks.	1	2	2	—	—	—	—	Penicillin should be given in large doses and may be combined with sulphadiazine.
39	Acute amœbic dysentery ..	—	—	1	1	—	—	—	—	
40	Leptospirosis (Weil's disease) ..	—	1	2	—	—	—	—	—	
41	Diphtheria (see remarks) ..	—	1	2	2	—	—	—	—	Antiserum essential.
42	*Herpes zoster*	—	—	1	—	2	—	—	—	
43	Tetanus (see remarks) ..	—	1	2	2	—	—	2	3	Antiserum essential.

NOTE.—(*a*) Whenever possible, sensitivity tests should be carried out and the appropriate antibiotic used. (*b*) While combinations of some antibiotics are often useful as indicated in the work-sheet, penicillin, which is bactericidal, should not be given with aureomycin, terramycin or chloramphenicol, as these antibiotics tend to interfere with the action of penicillin. (*c*) The importance of the development of antibiotic-resistant strains of organisms, especially in hospitals, is stressed. (*d*) Two new antibiotics have recently become available: erythromycin ("Ilotycin", "Erythrosin") and carbomycin ("Magnamycin"). Erythromycin is effective chiefly against the Gram-positive bacteria, its spectrum of activity being similar to that of penicillin. It is also active against *Hæmophilus pertussis*. "Magnamycin" is principally active against the Gram-positive bacteria with little or no action against Gram-negative bacteria. Its spectrum of activity also resembles that of penicillin. It has been shown that organisms will develop resistance to both these antibiotics. No cross-resistance with other antibiotics has as yet been demonstrated. It is felt that the use of these new antibiotics should be limited to the treatment of infections caused by organisms which are resistant to all other antibiotics. This restricted use will prolong the period of maximum value of these new antibiotics and should delay the appearance of resistant strains of organisms. (*e*) Polymyxin has not been included in the work-sheet, as it is a highly toxic antibiotic and should be used only under careful supervision with the patient in hospital. Polymyxin is effective against some strains of *Pseudomonas pyocyanea*. (*f*) Bacitracin is not included in the work-sheet, as the product at present available is suitable for local application only. (*g*) Neomycin, because of its toxicity, has not been included. The toxicity of viomycin ("Viocin") when administered twice a week is being investigated.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Annual Subscription Course.

Overseas Lecturer.

PROFESSOR J. GARFIELD DUNCAN, M.D., C.M., F.A.C.P., Professor of Clinical Medicine, Jefferson Medical College of Philadelphia, Pennsylvania, United States of America, and author of the well-known book "Diseases of Metabolism", will speak on "The Management of Essential Hypertension" on Tuesday, September 1, 1953, at 8.15 p.m., in the Stawell Hall, 145 Macquarie Street, Sydney.

Lectures on "The Scientific Basis of Medical Practice".

The following course of evening lectures under the general title of "The Scientific Basis of Medical Practice" has been arranged for members of the annual subscription course. All lectures will be held in the Stawell Hall, 145 Macquarie Street, Sydney, at 8.15 p.m.

Friday, September 4: "Fluid Distribution in Injury", Dr. F. C. Courtice, Director of the Kanematsu Memorial Institute of Pathology, Sydney Hospital. Wednesday, September 9: "The Cause of Cancer", Professor F. S. Magarey, Professor of Pathology in the University of Sydney. Tuesday, September 15: "The Physics of Isotopes as Applied to Medicine", Professor H. Messel, Professor of Physics in the University of Sydney. Friday, September 25: "Synaptic and Neuromuscular Transmission", Dr. Peter Bishop, Member of the Brain Research Unit in the University of Sydney. Friday, October 2: "Chemical Abnormalities of the Red Cell", Dr. C. R. Bickerton Blackburn, Director of the Clinical Research Unit, Royal Prince Alfred Hospital. Thursday, October 8: "Current Aspects of Enzymology", Professor J. L. Still, McCaughey Professor of Biochemistry in the University of Sydney.

The annual subscription course covers attendance at lectures by overseas lecturers and some specially arranged activities. Annual subscription is £2 2s. from July 1 to June 30. Fee for resident medical officers is £1 1s. Further details may be obtained on application to the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 5238, BW 7483. Telegraphic address: "Postgrad, Sydney."

Nominations and Elections.

THE undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Bentley, Alan, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952), 18 Anglo Avenue, Parkside, South Australia.

Brentnall, George Rex, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952), 92 Lockwood Road, Burnside, South Australia.

Hoff, Lothar Clemens, M.B., B.S., 1953 (Univ. Adelaide), 108 First Avenue, Joslin, South Australia.

Linn, John Malcolm, M.B., B.S., 1953 (Univ. Adelaide), Mallala, South Australia.

Nankivell, Shirley Dawn, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952), Box 58 Minlaton, South Australia.

Pfitzner, Murray Aikland, M.B., B.S., 1953 (Univ. Adelaide), Curramulka, South Australia.

Smith, Rayner Whitmore, M.B., B.S., 1953 (Univ. Adelaide), 46 Myall Avenue, Kensington Gardens, South Australia.

Whitehouse, Joseph Chenoweth, M.B., B.S., 1953 (Univ. Adelaide), 18 Albert Street, Goodwood Park, South Australia.

Wighton, Dugald Craven, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952), 96 Kingston Terrace, North Adelaide, South Australia.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JULY 18, 1953.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	6(3)	..	..	..	..	..	..	6
Amœbiasis	..	..	..	1(1)	..	..	..	..	1
Ancylostomiasis	..	..	1	..	..	..	1	..	2
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	1	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	6(6)	4(4)	10(10)	..	..	..	2	..	22
Diphtheria	5(3)	2(1)	18(4)	..	2(2)	2(1)	..	..	29
Dysentery (Bacillary)	..	1(1)	..	2(1)	..	..	..	..	3
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	13(7)	..	..	11(6)	..	..	..	24
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	3	..	..	..	..	..	3
Malaria	..	..	..	..	..	..	1	..	1
Meningococcal Infection	5(1)	1	2(1)	..	1	..	..	..	9
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	10(6)	1	2	3(2)	1	1	..	..	18
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	27(14)	..	..	21(17)	..	..	..	48
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	14(8)	57(38)	1(1)	..	2(2)	1	..	..	75
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	43(36)	15(7)	12(6)	4(3)	12(8)	3(1)	..	..	89
Typhoid Fever	2(1)	5(5)	1(1)	1(1)	1(1)	2(2)	..	..	12
Typhus (Flea-, Mite- and Tick-borne)	1	..	2	..	1(1)	..	..	..	4
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association: Moffatt, Cathie Douglas, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1953); Moffatt, Jeannie May Douglas, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1953); Marshman, Ian, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1953); Tymons, Frances Teresa, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Robertson, Thorburn Stirling Brailsford, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952).

Deaths.

THE following deaths have been announced:

MARTIN.—Lincoln William Martin, on August 9, 1953, at Victoria Park, Western Australia.

STOTT.—Ronald Gurney Stott, on August 2, 1953, at Kooyong, Victoria.

Medical Appointments.

Dr. G. S. Procopis has been appointed medical superintendent of State hospitals and homes in the Department of Public Health of New South Wales.

Dr. T. W. Miles has been appointed to the School Medical Service in the Department of Public Health of New South Wales.

Dr. O. W. Frewin has been appointed honorary medical officer in charge of the Geriatric Department at Northfield Wards, Royal Adelaide Hospital.

Dr. H. McIntyre Birch has been appointed Deputy Director-General of Medical Services, pursuant to the provisions of the *Mental Defectives Act*, 1935-1950, during the absence on leave of the Director-General of Medical Services of South Australia.

Dr. T. M. Greenaway has been appointed a government representative on the Board of Directors of the Royal Prince Alfred Hospital of New South Wales.

Dr. B. G. Wade and Dr. L. J. Woodland have been appointed members of the Physiotherapists Registration Board of New South Wales.

Dr. K. McL. Benn has been appointed medical officer in the Mental Hygiene Branch of the Department of Health of Victoria.

Dr. E. J. Hodder has been appointed public vaccinator to the Shire of Metcalfe, Victoria.

Dr. R. Hunter and Dr. M. K. Smith have been appointed senior surgical registrars at the Royal Adelaide Hospital in the Hospitals Department of South Australia.

The following part-time medical officers have been appointed at the Chest Clinic and Frome Ward, Adelaide, South Australia: physicians, Dr. J. G. Sleeman, Dr. A. C. Savage; assistant physician, Dr. R. C. Angove; clinical assistants, Dr. J. F. Jackson, Dr. J. M. M. Gunson, Dr. A. A. Jessup; orthopædic surgeon, Dr. E. F. West; general surgeon, Dr. O. W. Leitch; honorary laryngologist, Dr. P. G. Jay; thoracic surgery anæsthetists, Dr. J. E. Barker, Dr. J. A. Ferris, Dr. J. H. Stace.

Dr. R. R. Webb has been appointed deputy superintendent of the Medical Hospital and Receiving House, Royal Park, Victoria.

Dr. Colin William Phillips has been appointed quarantine officer at Port Adelaide, South Australia, under the provisions of the *Quarantine Act*, 1908-1950.

The following have been appointed to the honorary medical staff of the Royal Alexandra Hospital for Children, Camperdown, New South Wales: honorary assistant physician, Dr. B. T. Dowd; honorary relieving assistant physician, Dr. A. R. Tink; honorary dermatologist, Dr. R. F. A. Becke; honorary assistant dermatologists, Dr. M. B. Lewis and Dr. C. L. Statham; honorary relieving assistant dermatologist, Dr. Jean Mason-Johnson; honorary assistant ophthalmic surgeons, Dr. G. C. T. Burfitt-Williams and Dr. J. W. Hornbrook.

Dr. M. J. R. Drew has been appointed medical registrar at the Royal Adelaide Hospital.

Dr. J. H. Lindell has been appointed a member and chairman of the Hospitals and Charities Commission of the Department of Health of Victoria for a period of five years, pursuant to the provisions of the *Hospitals and Charities Act*, 1948.

Dr. Kenneth McGowen Doust and Dr. Joseph Arthur Robilliard have been appointed quarantine officers at Redland Bay and Port Kembla respectively under the provisions of the *Quarantine Act*, 1908-1950.

Professor H. R. Dew has been appointed a director on the board of the Prince Henry Hospital on the nomination of the Senate of the University of Sydney.

Dr. C. O. Cramp has been appointed a medical officer in the School Medical Service, Department of Public Health, New South Wales.

Dr. F. J. Kyneur has been appointed to the Division of Mental Hygiene, Department of Public Health, New South Wales.

Dr. M. H. Gabriel has been appointed medical superintendent of the Lazaret, Peel Island, Queensland.

Dr. J. A. Hede has been appointed medical superintendent of the Mental Hospital, Ipswich, Queensland.

Dr. R. A. Atherton has been appointed deputy medical superintendent of the Mental Hospital, Toowoomba, Queensland.

Diary for the Month.

AUG. 22.—Queensland Branch, B.M.A.: Annual General Meeting.
AUG. 25.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 26.—Victorian Branch, B.M.A.: Branch Council.
AUG. 27.—New South Wales Branch, B.M.A.: Branch Meeting.
AUG. 28.—Queensland Branch, B.M.A.: Bancroft Oration.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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